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SPONTANE SUBARACHNOÏDE BLOEDINGS (SUBARACHNOID HAEMORRHAGE)*

(DEEL I: DIE KLINIESE BEELD IN 30 GEVALLE)

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'En vir sy vader gesê: My kop, my kop! Dié sê toe vir 'n dienaar: Dra hom na sy moeder. En hy het hom na sy moeder gebring. En hy het op haar knieë gesit tot die middag en gesterwe.'

II Konings 4 : 19-20.

Spontane subarachnoïede bloedings het gedurende die twintiger jare as kliniese entiteit geleidelik uit 'n heterogene groep toestande te voorskyn getree wat almal vroeër as ‚apopleksie' bestempel is. Na die verskyning van Collier[1] se klassieke beskrywing in 1922 is dit as kliniese entiteit erken. Aangesien geen spesifieke vorm van terapie beskikbaar was nie, wat ook al die onderliggende patologie was, is 'n studie van etiologiese faktore as van suiwer akademiese belang beskou.[2] Die ontwikkeling van neuro-chirurgiese tegniek en serebrale angiografie regverdig nou egter 'n meer positiewe optrede by sulke pasiënte. Die vinnig groeiende literatuur op hierdie gebied is baie volledig deur Walton[3] hersien.

Die Suid-Afrikaanse literatuur oor hierdie onderwerp is uiters beperk. Geerling[4] (1933) noem subarachnoïede bloedings as 'n komplikasie van trauma. In 1944 beskryf Stein[5] 'n geval in 'n 9-jarige kleurlingdogter met 'n subakute bakteriële endokarditis. Appleton[6] (1948) beskryf die kliniese beeld en verloop van 'n pasiënt met 'n serebrale aneurisma wat oorlede is ten gevolge van so 'n bloeding. Gevalle met 'n subdurale hematoom, wat ook voldoen aan die criteria vir spontane subarachnoïde bloedings, word genoem deur Erasmus.[7] Allen[8] verwys na die chirurgiese behandeling in gevalle met subarachnoïede bloedings.

Die huidige werk beskryf die bevindings by 'n reeks van 30 gevalle met spontane subarachnoïde bloedings, toegelaat tot die Departement van Interne Geneeskunde by die Karl Bremer-Hospitaal. Die studie strek oor 'n periode van 2 jaar vanaf 1 Julie 1956 tot 1 Julie 1958. Slegs die kliniese beeld word bespreek in hierdie artikel. Ander waarnemings en gevolgtrekkings word nog vir publikasie voorberei.

Materiaal

Die 30 gevalle bestaan uit 12 Blankes en 18 Nie-blankes. Die Nie-blanke groep bestaan uit 14 Kleurlinge en 4 Bantoepasiënte. Die term, spontane subarachnoïede bloeding, is gebruik vir die gevalle waar die beeld, te wyte aan die intrede van bloed in die subarachnoïede ruimte, 'n primêre gebeure is. Dit sluit dus gevalle uit waar die beeld sekondêr tot trauma en neoplasme is.

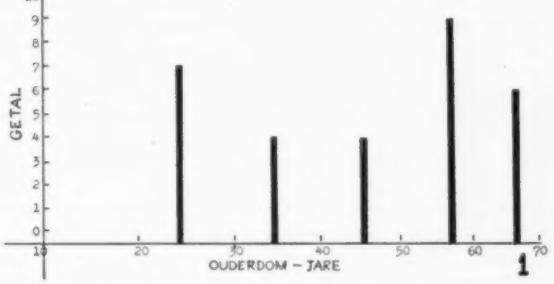
* Lesing gelewer tydens die tweede jaardag van die Mediese Skool van die Universiteit van Stellenbosch en die Karl Bremer-Hospitaal, Bellville, op 5 September 1958.

Die diagnose is in al die gevalle, op twee na, gemaak op grond van die kliniese beeld en bevestig met die bevinding van bloed en/of xantokromie in die serebro-spinale vog. Die 2 uitsonderinge is gediagnoseer op 'n geheel ‚tipiese' kliniese beeld, maar wat een week na die ictus helder vog by lumbaalpunksie getoon het in een geval, en 'n subhialoïede bloeding kenmerkend van subarachnoïede bloeding[3a] in die ander. Die criteria van McMenemey[9] is gebruik om te onderskei tussen 'n ware subarachnoïede bloeding en traumatiese lumbaalpunksie.

KLINIESE BEELD

Voorkoms. Gedurende die tydperk van studie was die aantal toelatings tot hierdie hospitaal 14,074. Hiervan is 2,932 tot die departement van interne geneeskunde toegelaat. Die voorkoms per 1,000 hospitaaltoelatings is dus 2·12; en per 1,000 gevalle tot die interne afdeling toegelaat, was daar 10·25 gevalle met subarachnoïede bloedings. Van die Blanke toelatings was 4 mans en 8 vrouens, en die Nie-blanke groep bevat 8 mans en 10 vrouens, wat die totale verhouding van mans tot vrouens in die verhouding 2 : 3 bring.

Ouderdom. Die ouderdomme varieer van 22 jaar tot 69 jaar, en toon 'n dubbele piek, nl. tussen 20-30 en tussen 50-60 jaar (Afb. 1).



Afb. 1. Ouderdomsdistribusie by 30 pasiënte met spontane subarachnoïde bloedings.

Seisoensvoorkoms. Geen definitiewe patroon is opgelet nie, en die verspreiding was ongeveer eweredig met 'n geringe styging in die lentemaande.

Geskiedenis voor die toeval. In nagenoeg die helfte van die gevalle (43·3%) is 'n geskiedenis van aanvalle van hoofpyn in die verlede gevind wat baie varieer in aard en distribusie. In 2 gevalle is 'n vorige geskiedenis van migraine gevind,

en in een van hierdie gevalle is die vader aan dieselfde soort hoofpyn onderhewig. Slegs in een geval kon 'n geskiedenis van 'n vorige bloeding gevind word. Onder die hipertensiegroep was slegs 2 bewus van hipertensie voor die aanval.

Swangerskap was in een geval aanwesig tydens die ictus (4 maande), terwyl een geval 4 maande, en 'n ander 16 maande na haar bevalling met 'n subarachnoïede bloeding presenteer het. Een geval het 7 swangerskappe gehad, maar het geen geskiedenis van 'n vorige bloeding nie.

Geassosieerde toestande. Een pasiënt is etlike jare lank al 'n bekende lyer aan diabetes mellitus wat met dieet alleen bevredigend gekontroleer is. In 'n ander pasiënt is littekens van epidermolysis bullosa gevind en die diagnose van porfirie is gemaak en bevestig deur die bevinding van 'n oormaat porfirien in die urine. Hierdie pasiënt is oorlede en by outopsie is 'n pulmonale embolis as oorsaak van dood gevind.

'n Geval met acanthosis nigricans en 'n geval wat met meningitis toegelaat is, en later 'n subarachnoïede bloeding ontwikkel het, is ook gevind. (Laasgenoemde word later meer volledig gerapporteer.)

Kardiovaskulêre toestande. Een pasiënt was op langtermyn antistollingsmiddels na 'n miokardiale infarksie en in onomkeerbare hartversaking toe hy 'n subarachnoïede bloeding ontwikkel het. 'n Geval met asimptomatiese aorta-insuffisiënsie is gevind. Sy Wassermann-reaksie was negatief, maar hy het penisillien-inspuitings vir 'n septiese voet gekry 'n paar maande van tevore. Geen tekens van subakute bakteriële endokarditis kon gevind word nie.

Geskiedenis van huidige aanval. Met een uitsondering, was die aanvang in al die gevalle skielik. Hoofpyn was die mees algemeen voorkomende simptoom en is in 76·6% van gevalle gevind. Slegs 4 kon die hoofpyn spesifiek lokaliseer terwyl die meerderheid van 'n ‚erge' of ‚barstende' hoofpyn gekla het.

In 63·3% van gevalle was daar bewussynsverlies wat in tydsduur gevarieer het van enkele minute tot koma waarmee die pasiënt toegelaat is en waarin hy oorlede is sonder om sy bewussyn te herwin. Van die gevalle het 10% konvulsies gehad en hieronder is ingesluit die bekende epilepsielyer wat in status epilepticus toegelaat is, met 'n bewese meningitis en wat later in die saal 'n bloeding ontwikkel het.

Nagenoeg die helfte van die pasiënte het 'n geskiedenis van vomering gehad by toelating, maar dié simptoom het in geen geval langer geduur as enkele ure na toelating nie. Twee gevalle het hematemese gehad na die ictus en is binne 48 uur na toelating oorlede.

Geestesverwardheid het in 43·3% van gevalle voorgekom, terwyl 2 gevalle volkome psigotiese gedrag getoon het by opname. Die psigose was 'n amnesties-konfabulatoriese simptoomkompleks soos by 'n Korsakoff-sindroom gevind word. Albei die psigotiese pasiënte het tydens hulle hospitaalverblyf na volkome normaal teruggekeer. In 20% van gevalle was daar 'n retrograde amnesie wat ook volkome herstel het. In 20% van gevalle was nekstyfheid 'n bewuste klagte terwyl 16·6% ook van fotofobie gekla het. (Dit sluit nie die geval met meningitis in nie.) Oormatige sweet is deur 13·7% van gevalle spontaan genoem en was ook veral 'n prominente klagte by die geval met 'n spinale subarachnoïede bloeding. Twee pasiënte het klagte van diplopie gehad en by albei kon 'n aneurisma as oorsaak van bloeding gedemonstreer word.

Bilaterale sciatica tydens die herstelperiode is eerste deur Arthur Hall[10] in 1932 beskryf, en hy het voorgestel dat dit te wyte is aan prikkeling van die cauda equina as gevolg van bloed in die subarachnoïede ruimte. Dit word dan soms ook as die Arthur Hall-sindroom bestempel. In ons reeks was daar een so 'n geval wat egter spoedig volkome opgeklaar het. Die simptome is getabuleer in Tabel I.

TABEL I. SIMPTOME BY 30 PASIËNTE MET SPONTANE SUBARACHNOÏEDE BLOEDINGS

	Totaal	*Persentasie*
Vorige hoofpyn	13	43·3
Migraine	2	6·7
Huidige geskiedenis:		
Hoofpyn:	23	
Frontaal	3	
Oksipitaal	1	76·7
Barstens, maar nie lokaliseerbaar	19	
Bewussynsverlies	19	63·3
Vomering	13	43·3
Verwardheid	13	43·3
Klagte van nekstyfheid	6	20·0
Amnesie	6	20·0
Fotofobie	5	16·6
Oormatige sweet	4	13·2
Duiseligheid	3	10·0
Konvulsies	3	10·0
Diplopie	2	6·6
Psigose	2	6·6
Hematemese	2	6·6

FISIESE BEVINDINGS

Die fisiese bevindings kan verdeel word in 5 groepe (Walton[3b]) en sal dus beskryf word onder: (1) Psigologiese effekte, (2) Tekens van konstitusionele respons, (3) Meningeale prikkeling, (4) Oogtekens en (5) Neurologiese tekens.

1. *Die geestestoestand* was normaal in slegs 30% van gevalle en dit omvat die meeste pasiënte wie se ictus etlike dae voor opname was. In Tabel II word die geestestoestand, soos gevind in hierdie reeks, in orde van voorkoms getabuleer. Dit varieer van buitengewone lomerigheid tot duidelike psigose of koma.

TABEL II. FISIESE TEKENS BY 30 PASIËNTE MET SPONTANE SUBARACHNOÏEDE BLOEDINGS

Geestestoestand	*Totaal*	*Persentasie*
Normaal	9	30
Gedisoriënteerd en lusteloos	7	23·3
Koma	4	13·2
Lomerig	3	10·0
Suf en apaties	3	10·0
Stuporeus	2	6·6
Psigoties	2	6·6

2. *Die konstitusionele respons* word weerspieël in die pols, bloeddruk, asemhaling en temperatuur, en in die urine deur albuminurie en glukosurie, terwyl die witbloedseltelling en besinking die hematologiese respons toon.

Die polsspoed varieer van 'n bradikardie tot tachikardie, maar die meerderheid val binne normale perke (Tabel III).

TABEL III. POLSSPOED

Spoed/minuut	30/60	60/70	70/80	80/90	90/100	100/110	110+
Getal	3	7	6	10	2	—	2

Die bloeddruk (Tabel IV) dui daarop dat die pasiënte in 3 groepe verdeel kan word. Die grootste groep is die normotensiewe groep. Ses gevalle was hipertensief en het so gebly. 'n Derde groep toon by opname, en oor die eerste paar dae na opname, 'n hipertensiewe lesing, maar dit keer terug na 'n normotensiewe lesing en bly dan deurgaans so.

Van die pasiënte met hipertensie was daar twee met

TABEL IV. BLOEDDRUKBEPALINGS MM. HG BY 30 PASIËNTE MET SPONTANE SUBARACHNOÏEDE BLOEDINGS

Normotensiewe groep	*Hipertensief, herstel na normotensie*	*Hipertensief, blywend*
120/80	230/110–160/90	180/100
115/80	170/100–160/80	245/120
105/60	200/105–130/80	175/100
140/100	220/140–130/90	200/130
120/70	170/120–130/80	150/110
135/80	230/120–140/90	220/100
115/70	240/140–150/80	
170/80		
110/80		
130/80		
170/60		
110/70		
190/80		
130/70		
130/90		
120/60		
120/60		
Totaal 17	7	6

papiledeem en hipertensiewe retinopatie wat egter presenteer het met 'n spontane subarachnoïede bloeding.

Die temperatuurkaart toon 'n groot variasie in patroon en feitlik elke soort koorskaart is gevind, van 'n blywende subnormale temperatuur tot 'n swaaiende temperatuur. In sommige gevalle was 'n byna normale temperatuur aanwesig met vinnige styging as terminale verskynsel. In ander gevalle het terminale hiperpireksie 'n skielike daling getoon. Die asemhaling is slegs van belang in soverre dat 3 pasiënte met Cheyne Stokes-asemhaling binne 48-uur na opname oorlede is. Die graad van albuminurie wissel van 'n ‚spoor' tot ‚2+' en is in 7 gevalle gevind. Die 3 met ‚2+' albuminurie was almal normotensief.

Glukosurie is in 6 gevalle gevind. Een geval met die bekende diabetes mellitus, wat net met dieet gekontroleer word, het by toelating 'n groen reduksie getoon. Die ander het geen geskiedenis van diabetes gehad nie en die reduksie was 'n verbygaande bevinding. Twee gevalle toon by opname 'n ‚baksteen'-reduksie. Een is in koma toegelaat met 'n bloedsuikervlak van 330 mg.% en is oorlede. Die ander het by opname 'n bloedsuikervlak van 167 mg.% gehad en het herstel met geen tekens van diabetes na die herstel nie.

Die witbloedseltelling varieer van normaal tot so hoog as 25,000 per c.mm. in een geval (Tabel V), terwyl die besinking normaal was in 6 pasiënte, maar in een geval selfs tot 100 mm. in die eerste uur was.

TABEL V. WITBLOEDSELTELLING EN BESINKING

Witbloedselle (c.mm.)	4,000–10,000	10,000–15,000	15,000–20,000	20,000+
27 Pasiënte	13	9	4	1 (25,000)

ROOIBLOEDSEL-BESINKINGSNELHEID. (WESTERGREN) MM./DIE EERSTE UUR (28 PASIËNTE)

Besinking	10	11–20	21–30	31–40	41–50	51–60	61–70	71–80	81–90	91–100
Pasiënte	6	4	4	3	2	4	1	1	2	1

3. Tekens van meningeale prikkeling. Die psigologiese bevindings, waarna reeds verwys is, vorm ook 'n deel van die beeld van meningeale prikkeling, nl. die rusteloosheid, die opgekrulde houding en die neiging om ondersoek te weerstaan, die komberse terug te trek, ens. Nekstyfheid (en Brudzinski se teken) en Kernig se teken is positief. In die geval met 'n spinale subarachnoïede bloeding het nekstyfheid eers 3 dae na die ictus gevolg, terwyl in 6 gevalle (20%) nekstyfheid afwesig was by opname. Die tyd van toelating na die ictus is van belang en word in Tabel VI weergee. Let op dat een geval slegs 'n paar uur na die ictus toegelaat is en nooit enige nekstyfheid getoon het nie. Nekstyfheid is soms gevind sonder 'n positiewe Kernig-teken en soos Walton[3c] vermeld, is eersgenoemde die mees betroubare teken van meningeale prikkeling. Die fotofobie in 5 gevalle is reeds genoem. Hoewel nie spesifiek vir subarachnoïede bloeding nie, is hierdie tekens die mees konstante en mees algemene fisiese bevindings van gevalle met subarachnoïede bloedings.

TABEL VI. NEKSTYFHEID BY 30 PASIËNTE MET SPONTANE SUBARACHNOÏDE BLOEDINGS

Teenwoordig	24
Afwesig	6
Tyd na ictus	paar uur, 3 dae, 4 dae, 7 dae, 21 dae en 28 dae.

4. *Oogtekens—retinale bloedings.* Subhialoïede bloedings is eerste deur Hale-White[11] in 1895 beskryf en is spesifiek vir 'n subarachnoïede bloeding. Sulke bloedings is in 4 gevalle gevind. In een pasiënt was dit bilateraal terwyl dit in 'n ander die diagnose bevestig het in die teenwoordigheid van normale serebro-spinale vogbevindings. Sulke bloedings kan binne 10 minute ontwikkel (Dandy[12]) terwyl Walton[3d] die ontstaan van so 'n bloeding kon waarneem. Ander tipes retinale bloeding kan voorkom en in 2 gevalle met hipertensiewe retinopatie is sulke bloedings gevind.

Papiledeem is gevind in 4 gevalle en in 2 hiervan maak dit deel uit van 'n hipertensiewe retinopatie. Die ander geval is die pasiënt met meningitis by wie die papiledeem verskyn het na sy 'n subarachnoïede bloeding in die saal ontwikkel het.

5. *Neurologiese tekens.* Die meeste skrywers, volgens Walton,[3e] meen dat in meer as 50% gevalle met subarachnoïede bloeding van aneurismale oorsprong (en selfs hoër in gevalle met angiomata), daar 'n mate van bloeding of vernietiging van serebrale weefsel teenwoordig is. Neurologiese tekens word in baie gevalle gevind, tog nie in almal nie, terwyl andersyds neurologiese tekens gevind kan word in persone sonder enige bewys van intraserebrale bloeding. Die neurologiese bevindings word in Tabel VII getabuleer. Ons het by 12 pasiënte uit die 30, neurologiese afwykings by ondersoek gevind.

Spinale subarachnoïede bloedings is seldsaam en in Walton se reeks was 3 gevalle (1%) te wyte aan so 'n bloeding.[3n] Die intrede van bloed in die subarachnoïede ruimte om die rugmurg gee aanleiding tot 'n skielike pyn soos 'n dolksteek, *coup de poignard*, van Mignon, en soos hierdie bloed opwaarts versprei, verskyn nekstyfheid en ander tekens van meningeale prikkeling. Die tydsverloop tussen die eerste verskynsel en laasgenoemde is afhanklik van die hoogte waar die bloeding in die spinale subarachnoïede ruimte plaasgevind het. Opistotonus is algemeen in hierdie gevalle. Retensie van urine is algemeen en die geweldige pyn mag aanleiding gee tot moeilikheid by die diagnose en abdominale katastrofe of nierkoliek mag foutiewelik diagnoseer word. In 2 gevalle in die literatuur beskryf, is 'n Dietl

TABEL VII. NEUROLOGIESE BEVINDINGS BY 30 PASIËNTE MET SPONTANE SUBARACHNOÏEDE BLOEDINGS

Pasiënt		*Kraniaalsenuwees*		*Hemiplegie-parese*		*Hemianopie*		*Pupilreaksie*		*Urine*	*Verloop*
Geslag	*Oud.*	*L.*	*R.*	*L.*	*R.*	*L.*	*R.*	*L.*	*R.*		
V	28			+		+	+				Blywend
V	22								Wyd.		
V	54									Retensie	Duur 3 weke
V	46			+							Dood
V	63	VII				+					
		XI, XII									
V	25	VI			+						Herstel
M	59		III								Blywend
M	40		VII								
M	±40		VII	+							
M	53			+							
V	25				+						Herstel
V	28				+						Herstel

se krisis gediagnoseer.[13] Die sciatica-soort pynverspreiding in die bene mag verwar word met 'n Arthur Hall-sindroom. Die derde groep simptome is verlammings te wyte aan beskadiging van die murg. Die bevindings by ons geval met 'n spinale subarachnoïede bloeding word kortliks weergee:

Blanke vrou, 54 jaar oud. Nefrektomie gehad vir ? abses van die regternier, 34 jaar gelede. Was volkome gesond tot 6 dae voor opname toe sy skielik gewek is deur 'n pyn ‚soos 'n messteek in die rug' (ongeveer op die hoogte van lumbaal 1-2). Drie dae later het sy 'n oksipitale hoofpyn ontwikkel en gevind dat haar nek styf en pynlik is. Sy sweet aanhoudend en buitengewoon baie sedert die aanval. Sedert die dag voor opname het sy geen urine passeer nie.

By ondersoek is gevind 'n vrou met opistotonus, nekstyfheid en 'n positiewe Kernig-teken. Die bloeddruk was 200/105 mm. Hg, maar herstel spontaan na 130/80 mm. Hg en bly konstant normotensief. Die blaas is tasbaar tot by die umbilikus en kateterdreinering met vloed-dreinering is toegepas. Die retensie van urine het 3 weke lank voortgeduur en het toe spontaan herstel. Angiografie (serebraal en vertebrale angiografie) en 'n miëlogram het geen afwykings getoon nie. Sy het volkome herstel.

BESPREKING

'n Oorsig van die Suid-Afrikaanse literatuur is reeds in die inleiding gegee. Die kliniese beeld sal waar toepaslik vergelyk word met bevindings van oorsese studies.

Die eerste opvallende bevinding is dat die toestand meer algemeen in hierdie hospitaal gesien is as in reekse deur Walton aangehaal. So byvoorbeeld, vind Taylor en Whitfield[14] (1936) 'n voorkoms van 0·63/1,000 hospitaaltoelatings en Walton [3f] se 312 gevalle toon 'n voorkoms van 1·7/1,000 hospitaaltoelatings (1956). Hy meen dus dat die toestand òf meer dikwels voorkom, òf meer dikwels gediagnoseer word as tydens Taylor en Whitfield[14] se studie. Ons syfer van 2·12/1,000 is aansienlik hoër as dié van Walton en dit mag wees dat die toestand meer algemeen is in Suid-Afrika.

Walton,[3g] haal 4 reekse[14-17] aan wat geen verskil in die geslagsvoorkoms toon nie en 3 reekse[18-20] wat 'n hoër voorkoms onder vrouens toon. Hy wys daarop dat slegs een klein reeks[21] meer mans as vrouens toon. Hy kom tot die gevolgtrekking dat die toestand ietwat meer dikwels onder vrouens voorkom en ons bevinding van 'n 2 : 3 verhouding, mans tot vrouens is in ooreenstemming hiermee.

Die dubbele piek in ouderdomsvoorkoms, meen ons, is toe te skryf aan die twee etiologiese toestande: kongenitale vaskulêre afwykings, bv. aneurismas en angiomata in jonger mense en degeneratiewe veranderings in die ouer groep pasiënte, maar ons bevindings strook met dié van ander reekse wat die maksimum voorkoms tussen 40 en 60 jaar vind.

Die hoë voorkoms van vorige hoofpyne in hierdie reeks is waarskynlik te wyte aan die feit dat dit 'n prospektiewe studie was en daar noukeurig gelet is op die geskiedenis voor die aanval. Wat migraine betref, is die posisie deur Frankel[22] hersien en hy dui daarop dat in die verwantskap van die toestand tot subarachnoïede bloedings dit belangrik is om te probeer bepaal of die migrainehoofpyn wat in kinderjare begin, die eerste simptoom is van 'n aneurisma, of hulle toevallig geassosieerde toestande is en of die migraine die ontstaan van die aneurisma bevorder. Walton[3h] haal skrywers aan wat bewyse ten gunste van al 3 hierdie moontlikhede lewer.

Die geassosieerde toestande wat in ons reeks voorkom is divers en 'n spesifieke assosiasie is nie gevind nie. Die verhouding tot swangerskap is egter van belang. Die probleme by hantering van 'n geval met 'n subarachnoïede bloeding tydens swangerskap kan moeilik wees aangesien 'n subarachnoïede bloeding *per se* albuminurie en hipertensie kan veroorsaak en in 'n komateuse pasiënt kan die toestand maklik as eklampsie gediagnoseer word. Selfs as die diagnose van subarachnoïede bloeding bevestig is in so 'n geval, sal dit moeilik wees om toksemie as 'n bydraende faktor uit te skakel.[3i] Die feit dat 2 van ons pasiënte 4 en 16 maande na 'n bevalling met 'n subarachnoïede bloeding gepresenteer het, laat die vraag ontstaan of verhoogde intrakraniale druk tydens afdrukpogings 'n klein aneurisma so kon vergroot het dat dit tot 'n bloeding gelei het. Die ander geval wat egter 7 swangerskappe deurgemaak het, en jare later eers 'n bloeding ondervind het, weerspreek weer die effek van swangerskap op die vaskulêre defek. Behalwe vir 'n hoër voorkoms tydens swangerskap gekompliseer deur toksemie, as in normale swangerskap, kan daar volgens Walton[3i] nie 'n gesaghebbende opinie oor die verwantskap tussen swangerskap en subarachnoïede bloedings gegee word nie.

Die skielike aanvang en die prominensie van hoofpyn in die geskiedenis van subarachnoïede bloedings word allerweë erken. Die volgende prominente teken is skielike bewussynsverlies.[3j] Die feit dat 'n psigotiese beeld en konvulsies kan voorkom as aanvangsimptome is uiters belangrik in die differensiële diagnose en beklemtoon die belang van 'n deeglike fisiese ondersoek in gevalle wat as psigose of status epileptikus voordoen.

By fisiese ondersoek kan die bevindings baie uiteenlopend wees, maar tog is die kliniese beeld redelik kenmerkend, nl. 'n skielike aanvang met hoofpyn, bewusteloosheid of ander simptome soos genoem in die kliniese beskrywing. Die

bevinding van tekens van meningeale prikkeling, die belangrikste waarvan nekstyfheid is, sal noodwendig lei tot 'n lumbaalpunksie waarby die diagnose dan gefinaliseer kan word. Die konstitusionele tekens kan die beeld van 'n meningitis presies naboots.

Die moeilike gevalle is diegene sonder nekstyfheid en ander tekens van meningeale prikkeling, maar 'n skielike, erge hoofpyn regverdig altyd 'n lumbaalpunksie.

Dit is belangrik om in gedagte te hou dat 'n redelik massiewe glukosurie of albuminurie kan voorkom en in 'n komateuse pasiënt, miskien met asidose as gevolg van uithongering en/of braking, kan dit lei tot moeilike onderskeiding tussen 'n diabetiese koma en 'n bloeding. Die meningeale prikkeling is gewoonlik die onderskeidingsteken. (Die een geval met 'n baksteenreduksie en asetoon in die urine, is as diabetiese koma verwys. Die nekstyfheid het gelei tot lumbaalpunksie waar die diagnose van subarachnoïede bloeding gemaak is. Die familie kon ook vertel dat sy volkome gesond was tot 'n skielike hoofpynaanval voorgekom het gevolg deur koma kort daarna.)

Dit word dikwels beweer dat die pols stadig is in hierdie toestand vanweë verhoogde intrakraniale druk,[23] maar soos in Walton[3k] se reeks kon ons geen konstante verband vind nie. Bradikardie en tachikardie is gevind maar die meerderheid van gevalle het 'n normale polsspoed getoon.

Die bloeddruk in gevalle met 'n subarachnoïede bloeding moet opgevolg word. Verskeie skrywers het die tydelike styging in bloeddruk beklemtoon, maar in Walton se reeks was dit meer as eenmaal bepaal in slegs 13 gevalle. Die meganisme vir so 'n tydelike styging word toegeskryf aan die verhoogde intrakraniale druk en sy effek op die hipotalamiese senters.[3l] 'n Groot persentasie (20%) van ons gevalle toon so 'n tydelike styging van bloeddruk.

Albei die gevalle wat hematemese gehad het, is oorlede. Arteta[24] het gewys op die voorkoms van maagulkus wat deur prikkeling van die dele van die frontale korteks wat hipotalamiese aktiwiteit kontroleer, veroorsaak word. Die lokale letsel in die maag in gevalle met serebrale toestande met hematemese is onlangs beskryf deur Doig en Shafer.[25] Die essensiële letsel is skynbaar 'n veneuse kongestie met bloeding onder die mucosa en later met oppervlakkige ulserasie. Interessant is dus in hierdie verband die bevinding van 'n poliep in die maag, met 'n subakute ulkus daarnaas, vanwaar bloeding plaasgevind het in een van ons gevalle met hematemese.

Die essensiële meganisme van glukosurie word beskou as prikkeling van die hipotalamiese senters met mobilisasie van lewer-glikogeen deur simpatiese stimulering *via* die splanchniese senuwees.[3m] Meadows[26] beweer egter dat glukosurie ontstaan sonder hiperglisemie, 'n bevinding wat in ons 2 gevalle met 'n baksteenreduksie in die urine en verhoogde bloedsuiker nie bevestig kon word nie.

Lumbaalpunksie en sy belangrikheid in die diagnose van die toestand kan nie oorbeklemtoon word nie. In ons gevalle is lumbaalpunksie, waar moontlik, elke tweede dag gedoen en die bevindings hiervan sal elders gerapporteer word. In geen geval het ons 'n nadelige effek, toe te skryf aan die lumbaalpunksie, waargeneem nie. Dit is egter belangrik dat 'n minimum vog deur 'n dun naald geneem word. Manometriese bepalings moet geskied sonder die Queckenstedt-maneuwer. As 1-2 ml. vog verwyder word, is lumbaalpunksie in hierdie gevalle veilig selfs as daar verhoogde intrakraniale druk is.[2]

Die mortaliteit, sover onmiddellike prognose betref, (d.w.s. tydens die pasiënt se hospitaalverblyf) bevind ons 26·8% in ons reeks, maar dit word ook elders volledig bespreek.

OPSOMMING

1. 'n Prospektiewe studie van gevalle met spontane subarachnoïede bloeding is by die Karl Bremer-Hospitaal gemaak. Dertig gevalle is oor 'n tydperk van 2 jaar nagegaan.

2. Die kliniese bevindings is noukeurig nagegaan en waarnemings oor die simptome, tekens en gewone laboratorium-ondersoeke word hier uiteengesit.

3. Die voorkoms van 2·12 per 1,000 hospitaaltoelatings is hoër as die syfer in oorsese reekse en dit word voorgestel dat die toestand miskien meer algemeen in Suid-Afrika is as oorsee.

4. Daar word gewys op probleme by die differensiële diagnose, en huidige opvattings oor die meganisme van sommige tekens van die toestand word genoem.

5. 'n Geval met 'n spinale subarachnoïede bloeding word bespreek.

SUMMARY

1. A prospective study of patients presenting with spontaneous subarachnoid haemorrhage was conducted at the Karl Bremer Hospital. Thirty patients were examined over a 2-year period.

2. The clinical findings were studied in detail, and the observations on symptoms, signs and the usual laboratory procedures are presented here.

3. The incidence of 2·12 cases per 1,000 hospital admissions is higher than figures quoted for overseas series and it is suggested that the condition might occur more frequently in South Africa than abroad.

4. Problems in the differential diagnosis are discussed, and contemporary views on the mechanisms of some of the physical signs are mentioned.

5. A case of spinal subarachnoid haemorrhage is discussed.

Ons wil graag ons dank uitspreek aan die Superintendent van die Karl Bremer-Hospitaal vir verlof tot publikasie en aan mej. Y. Stuart vir die tikwerk verbonde aan die manuskrip.

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DIE ERFLIKHEID VAN POLIKISTEUSE NIERE*

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Dit is wenslik dat aan die begin van hierdie bespreking daarop gelet word wat presies verstaan word onder die begrip: Polikisteuse niere. Vanweë die insluiting van talle verskillende vorms van kisteuse niere in een groot groep, is die denke oor hierdie onderwerp eerder verwar as opgehelder. In hierdie werk sal ons onder polikisteuse niere slegs vergrote niere insluit waarvan die normale weefsel feitlik geheel-en-al verplaas is deur kiste van verskillende grootte en met 'n verskillende inhoud. Ten spyte van hierdie omvattende veranderinge behou die niere egter hulle karakteristieke niervorm. In die uitgesproke toestand is hierdie siekte nie moeilik om te onderskei van ander kisteuse toestande van die nier nie.

'n Ander basiese begrip wat ook duidelik gestel moet word is dat daar in hierdie werk 'n duidelike onderskeid gemaak word tussen polikisteuse niere by pasgebore kinders en by volwassenes. Die gronde waarop so 'n onderskeid gemaak word, sal nie in besonderhede bespreek word nie, maar die belangrikste verskilpunte is:

(*a*) Baie duidelike verskil in ouderdom waarop die siekte voorkom.

(*b*) In pasgebore en klein kinders met hierdie siekte is daar baie dikwels een of meer ander aangebore defekte teenwoordig. Soortgelyke defekte is in die volwassene met polikisteuse niere beslis skaars, en van meer beperkte aard.

(*c*) Die siekte toon in die twee vorms 'n duidelike verskillende wyse van oorerwing en kom nooit in dieselfde familie voor nie.

(*d*) Daar is histologiese verskille tussen die twee.

Voorkoms

Polikisteuse niere, in watter vorm ookal, is 'n skaars toestand. Nasporing van die frekwensie van voorkoms in verhandelinge oor die onderwerp toon 'n kliniese voorkoms wat wissel van 1 in 184 tot 1 in 6,515 toelatings. Die groter frekwensie in sekere skrywers se werk is toe te skryf aan die feit dat hulle gewoonlik vanuit urologiese klinieke skryf. Die kliniese voorkoms is egter geen juiste indeks van die voorkoms van hierdie siekte in die algemene bevolking nie. Baie persone wat die nierdefek het, ondervind daar geen simptome van nie, en andere het slegs simptome vanweë kompliserende faktore van die nierafwyking sonder dat die basiese toestand dan gediagnoseer word. Om hierdie redes is dit dan ook verstaanbaar dat polikisteuse niere baie meer dikwels voorkom in post mortem materiaal as in kliniese materiaal. Dit is belangrik om by te voeg dat die insluiting van doodgebore kinders en pasgebore kinders in 'n patologiese reeks ook tot 'n baie hoër voorkomsyfer lei.

Familiestamboom

Die gedagte om hierdie siektetoestand se erflikheid na te spoor het toevallig begin nadat 3 pasiënte met polikisteuse niere in algemene praktyk waargeneem is. Later is vasgestel dat die 3 pasiënte tot een familie behoort. 'n Volledige familiestamboom is opgestel en die ander lede van die familie is opgespoor en klinies en radiologies ondersoek. Soveel informasie as moontlik is ingewin oor dié familielede wat reeds oorlede is en ou hospitaalrekords is opgespoor ten einde die diagnose te bevestig sover dit moontlik was.

Tot op die huidige tydstip is 28 gevalle gevind van polikisteuse niere wat sonder enige twyfel bewys is. In 6 familielede wat reeds oorlede is, was die siekte waarskynlik teenwoordig—gegrond op 'n geskiedenis van groot niere en hematurie. In 12 lede van die familie is daar vroeë tekens van die siekte, maar nie so uitgesproke dat 'n onbetwisbare diagnose gemaak kan word nie. In hierdie familie word die gene vir polikisteuse niere deur vyf geslagte ge-erf. Nog twee kleiner families is opgespoor, maar kon nie so volledig ondersoek word nie. Al die afwykinge wat al beskryf is in kombinasie met polikisteuse niere by volwassenes is nagespoor en hierdie familie is daarvoor ondersoek ten einde vas te stel of daar enige genetiese koppeling kon aangetoon word. Met dieselfde doel voor oë is bloedgroepbepalings gedoen op 40 van die lede van die eerste familie. Daar kon geen verband gevind word tussen polikisteuse niere en ander defekte nie, behalwe serebrale aneurisma en kisteuse lewer soos reeds welbekend is.

Erflikheid

Uit hierdie families, en uit die families wat beskryf is in die wêreldliteratuur, kan ons sonder enige twyfel sê dat ons in die volwasse vorm van polikisteuse niere te doen het met 'n dominante vorm van oorerwing. Daar was geen ondertrouery in hierdie familie nie.

Vanweë die feit dat die siekte van vader of moeder oorgedra word aan kinders van beide geslagte in gelyke verhoudings, kan ons aanneem dat hier geen bewys is vir geslagsgekoppelde oorerwing nie.

Daar kon geen bewys gevind word dat die siekte in latere geslagte vroeër voorkom as in hul voorafgaande geslagte nie. 'n Betreklike groot aantal gevalle met die siekte is onder die ouderdom van 20 jaar gevind in die vyfde geslag, maar hulle was almal asimptomaties en is toevallig gevind omdat die hele familie ondersoek is. Dit is waarskynlik dat die meeste van hulle geen simptome sal hê vir 'n hele aantal jare nie.

Geen bewys kon gevind word dat die siekte 'n geslag oorslaan d.w.s. van 'n voorvader na 'n kleinkind gedra word sonder dat die draer tussen-in die siekte vertoon nie. In een geval lyk dit oënskynlik so, maar die informasie oor die draer is so beperk dat geen besliste uitspraak hieroor gegee kan word nie.

Indien onder die begrip van penetrasie verstaan word die frekwensie van manifestasie van 'n eienskap in persone wat die gene vir daardie eienskap dra, vind ons in polikisteuse niere feitlik 'n 100% penetrasie. Soms moet 'n persoon egter 'n betreklike hoë ouderdom bereik alvorens die siekte tot uiting kom en dan nog mag die draer daarvan sonder enige simptome wees.

Variasie in Ekspressie

Wanneer die kliniese eienskappe van polikisteuse niere beskou word, selfs in dieselfde familie, is daar 'n baie groot

* Opsomming van 'n lesing gelewer tydens die tweede jaardag van die Mediese Skool van die Universiteit van Stellenbosch en die Karl Bremer-Hospitaal, Bellville, op 5 September 1958.

verskil in die ouderdom waarop die siekte begin, die graad waarin dit tot uiting kom en die wyse waarop dit die pasiënt aantas. Dit wil voorkom dat hier bewyse gevind kan word vir erflikheid deur 'n gene wat 'n groot variasie in ekspressie toon.

Die redes hoekom aangeneem word dat daar 'n monogenetiese oorerwing met 'n varieerbare ekspressie verantwoordelik is vir hierdie siekte, liewers as 'n poligenetiese stelsel, sal nie hier volledig bespreek word nie.

Dit is interessant dat daar verskeie siektes is waarin daar kiste van een of ander aard in die nier teenwoordig is, tesame met ander ontwikkelingsabnormaliteite. In polikisteuse niere by volwassenes is daar dikwels kiste in die lewer, soms in die pankreas of milt, en betreklik dikwels ook serebrale aneurisma.

By polikisteuse niere in klein kinders is daar dikwels veelvuldige ander gebreke, maar veral ook kiste in die lewer. Soms word, as 'n saamgestelde afwyking wat in verskeie kinders kan voorkom, die volgende gevind: Polikisteuse niere, pankreas en lewer, meningocoel en polidaktilie met ander minder algemene afwykings soms nog hierby gevoeg. Von Hippel-Lindau se siekte is 'n ander toestand waarin veelvuldige nierkiste—maar nie polikisteuse niere, gevind word tesame met hemangioblastome van die sentrale senuweestelsel en dikwels ook kiste in die pankreas. Ook in tuberose sklerose word nierkiste gevind as 'n onderdeel van 'n baie wydverspreide siektetoestand. Slegs tweekeer, in pasgeborenes, is ware polikisteuse niere saam met tuberose sklerose beskryf.

Dit sou beslis onwys wees om al hierdie toestande in een groep te wou saamtrek alhoewel hulle, oppervlakkig beskou, miskien heelwat ooreenkoms mag toon. Dit is egter interessant en beslis belangrik, dat in verskillende, wyduiteenlopende toestande, kiste van nier, lewer en pankreas in verskillende kombinasies kan voorkom. Weereens stuit ons op die vraag: Het ons hier te doen met 'n enkel gene wat kisteuse toestand van die viscera beïnvloed, maar met 'n wye varieerbaarheid in ekspressie toon, of is dit hier 'n poligeniese sisteem wat verantwoordelik is? Alleen deur die versameling van soveel materiaal as moontlik en deur noukeurige analise daarvan kan mettertyd 'n begrip ontwikkel van die menslike genetiese struktuur, en hierdie vrae beantwoord word.

THEME OF THIS STUDY

This study of the heredity of polycystic kidneys was begun after 3 patients with polycystic kidneys had been examined in general practice. Later it was found that these 3 patients were members of the same family. A complete family tree was drawn up and the other members of the family were traced and examined clinically and radiologically. As much information as possible was obtained regarding those members of the family who had already died, and old hospital records were investigated in order to confirm the diagnosis.

Hierdie artikel is slegs 'n voorlopige verslag en as sulks ook net 'n uittreksel uit 'n groter werk oor die erflikheid en kliniese aard van polikisteuse niere wat later gepubliseer sal word. Alle literatuurverwysings en erkennings sal by die latere publikasie ingesluit word.

CAUSTIC COMMENT*

The inability of so many well qualified physicians to express themselves effectively was stated by Alan Gregg, Director of Medical Sciences of the Rockefeller Foundation in an address at Jefferson Medical College in 1943: 'The common level of medical and scientific writing in our professional books and journals already constitute the most serious internal limitations to medical education and research.'

Hewitt maintains that the report of research is part of the research and that the standards which the investigator requires of himself at the bedside, the operating table, and the laboratory bench, should be carried undiminished to the library, the study, and the desk.

Here are Hewitt's own words: 'To a responsible writer, the manuscript with his name on it, is the highest product of his mind and art; as nearly flawless as he can make it, his property, rooted in his character and evidence of it. He is, moreover, scrupulous in matters of credit, use of quotation marks, accuracy of quotation, accuracy of paraphrase and accuracy of reference. All this implies that he can, and will, read accurately.

'There was a time, and I lived in the latter years of it, when knowledge of the foregoing principles, and possession of the attendant abilities, were parts of the equipment of an educated man. However he might earn his living, if he had behind him a formal education, its foundation generally was in the humanities. Laying of that foundation, moreover, had begun early in his life.

'Only in 1941 did I realize that laying the foundations for skilful English composition had been further delayed when I read the following in an article by Professor Bredvold, of the University of Michigan: "Our High School graduates have no real working knowledge of grammar, and what is worse, they have acquired, somehow, a contempt for such trivial study . . . grammar and rhetoric are, like logic, abstract and formal subjects. Our freshmen are not intellectually ready for abstract and formal subjects."

'What then is the prospect for the sons or grandsons of us who are here today, when a few years hence, they are preparing to enter the medical school? Can they, in the little time they are allowed for the humanities in the premedical course—can they compensate for the foundation in abstract and formal subjects which they did not get in grade school and high school? Some can and will, of course. The majority cannot or will not *and, unless they obtain help somewhere* no matter how eminent they may become in their chosen fields, they will be likely not to command as much respect in the learned world as might have been theirs if they had been equipped, in abstract and formal thinking, to hold their own.'

* Extracted from an address by H. Swanberg, B.S., M.D., F.A.C.P., presented at the Symposium on Medical Writing, 9th annual meeting, American Medical Writers' Association, St. Louis, 1 October 1952.

VAN DIE REDAKSIE : EDITORIAL

AFRIKAANS AS MEDIESE VAKTAAL

Nadat ons by 'n vorige geleentheid[1] geskryf het oor die huidige stand van Afrikaans as mediese vaktaal, het daar 'n uitgebreide korrespondensie oor die saak gevolg. Ons korrespondente was veral begaan daaroor dat daar mettertyd aangetoon sou word watter vordering al gemaak is. Dit is dus waarskynlik nou 'n geskikte geleentheid om die hele probleem weer te stel en om vordering te rapporteer.

Aan die begin moet ons dit weereens herhaal dat die skepping van 'n heel nuwe mediese terminologie geen geringe taak is nie. Dit is nie 'n taak wat volgens 'n formule afgehandel kan word nie. Gestadige maar progressiewe groei is nodig. Ook dien dit vermeld te word dat die probleem waarvoor ons staan waarskynlik vandag enig van sy soort in die wêreld is—met die uitsondering miskien van die Staat van Israel, waar ook 'n nuwe mediese terminologie geskep moes word. Onder omstandighede soos dié waaronder ons werk, is daar 'n paar moontlike weë wat gevolg kan word.

Om mee te begin kan ons dit oorlaat aan persone wat in Afrikaans skryf om die soort gebruike te volg wat hul self verkies—met dien verstande dat daar 'n poging tot konsekwente skryfwyse moet wees.

Tweedens moet daar 'n aktiewe komitee of komitees geskep word met die bedoeling om die betrokke probleme sistematies te probeer benader.

Derdens moet samewerking tussen die twee Afrikaanse universiteite sowel as al die beoefenaars van mediese Afrikaans, sover moontlik, gesoek word.

Deur middel van die *Tydskrif* is ons reeds besig om uitvoering te gee aan die eerste aanbeveling, naamlik, om skrywers min of meer hul eie gebruik te laat volg. Uit hulle pogings leer ons en bou ons ons eie gebruike op.

Dan is daar 'n taalkomitee aangestel deur die Fakulteit van Geneeskunde van die Universiteit van Stellenbosch, met die doel om eenvormigheid te probeer verkry by die gebruik van mediese terme aan die Mediese Skool van die Universiteit van Stellenbosch. Hierdie komitee bestaan uit proff. F. D. du T. van Zijl, H. E. Brink, A. J. Brink, J. N. de Villiers, J. F. v. E. Kirsten, en W. Kempen. Die Universiteit van Stellenbosch het die Redakteur van die *Tydskrif* ook uitgenooi om op die komitee te dien. Die doel en strewe van hierdie komitee is om gereelde maandelikse byeenkomste te hou waarop lyste van probleemwoorde bespreek sal word. Enige lid van die Mediese Vereniging kan deel hê hierin deur probleme vir ondersoek en bespreking aan ons te stuur. Ons het reeds al lyste van sulke woorde ontvang. Tweedens is dit die doel van die komitee om sistematies te begin met die opstel van 'n woordeboek wat hopelik mettertyd 'n standaardwerk sal kan word. Derdens wil die komitee langs alle moontlike weë samewerking soek met dergelike groepe en komitees in die Noorde en elders.

Om op te som sou ons dus kon sê dat die genoemde komitee dit beoog, nie om Afrikaans as mediese vaktaal kunsmatig ryp te maak nie, maar om aktiewe en positiewe leiding te gee. Ons hoop verder om in die toekoms gereeld lyste van woorde te publiseer, op die grond van bereikte ooreenkomste, en om aanbevelings te maak insake gebruikswyses. Weereens wil ons almal wat in hierdie saak belangstel, uitnooi tot aktiewe ondersteuning en medewerking.

1. Van die Redaksie (1958): S. Afr. T. Geneesk., 32, 675.

ANTIEMETIC DRUGS

In recent years a number of drugs have been produced which, by their ability to depress specific areas of the brain, are effective in controlling vomiting caused by various conditions. These different agents vary in their site of action and their efficacy in controlling vomiting.

As recently as 1948, Borison and Wang first demonstrated an area in the lateral reticular formation which is very responsive to electrical stimulation. This centre is activated by a number of stimuli, metabolic as well as nervous, and does not itself initiate vomiting. Activation of this centre occurs *via* several pathways, for example through labyrinthine reflexes and visceral reflexes and the so-called chemoreceptor emetic trigger zone in the floor of the fourth ventricle (the area postrema). Thus apomorphine, which has long been classified as a central emetic, does not directly stimulate the vomiting centre, but stimulates the trigger zone which acts on the closely related centre through internuncial neurones. Antiemetic drugs, such as certain antihistamine drugs, e.g. cyclizine (marezine), diphenhydramine (benadryl), and dimenhydrinate (dramamine), and certain phenothiazine derivatives, e.g. chlorpromazine, act on the trigger zone as well as on other zones in the brain and brain stem. All these antiemetic drugs may produce side-effects such as sedation, dizziness, blurring of vision and other reactions which are usually mild and frequently dependent on the dosage. More serious toxic effects are sometimes produced, e.g. on the bone marrow and the liver by chlorpromazine.

Emesis can be produced by drugs stimulating the trigger zone or the vomiting centre itself, or by gastrointestinal irritation, causing reflex stimulation of the vomiting centre. Antiemetic drugs may block one or more of these mechanisms.

For the nausea and vomiting secondary to infection, hepatitis, cholecystitis, salpingitis, pneumonia, and urinary tract infection, chlorpromazine is a most effective agent.[1,2] In common infections occurring in children cyclizine has proved highly successful. Intestinal obstruction must, in all cases, be excluded before antiemetic drugs are administered. In uraemia, too, chlorpromazine may control vomiting and simplify the management of patients suffering from this disease. In radiation sickness chlorpromazine, diphen-

hydramine, and certain other drugs may be beneficial in many cases.

In postoperative vomiting the aetiology may be complex, for irritation of the alimentary canal, toxins, psychological factors, dehydration and acidosis may all stimulate the vomiting centre. In these cases chlorpromazine, diphenhydramine and dramamine (among others) have proved effective.[3] These drugs may be more valuable when given as a prophylactic measure—dramamine, followed in the postoperative period by chlorpromazine, for cases where vomiting occurs. On the other hand, chlorpromazine may be more valuable as a pre-anaesthetic agent for potentiation of anaesthesia.

During the nausea and vomiting of pregnancy meclizine (bonamine) has proved especially effective. This drug has the advantage of prolonged duration of action, but other drugs mentioned above have also been used with success in some instances.[4] In severe persistent vomiting chlorpromazine may be the drug of choice; however, sedation, orthostatic hypotension, and dizziness, may prove a disadvantage.

Chlorpromazine is very useful against drug-induced vomiting and in vomiting occurring in intoxications such as uraemia, diabetic acidosis, carcinomatosis, or radiation sickness. Diphenhydramine, meclizine, and cyclizine may be more effective in suppressing vomiting produced by analgesics (morphine, pethidine, methadone) because of their lower incidence and severity of side-effects; but if they are ineffective chlorpromazine should be tried. If a patient is unable to tolerate oral administration, intramuscular injection may be given, followed by oral medication.

Many other antiemetic agents related to the compounds already mentioned have been undergoing trial, the aim being to find a specific depressant of the vomiting mechanism which will have only minor effects on other systems.

1. Moyer, J. H., Kent, B., Knight, R. W., Morris, G., Dizon, M., Rogers, S. and Spurr, C. (1954): Amer. J. Med. Sci., 228, 174.
2. Moyer, J. H., Kinross-Wright, V. and Finney, R. M. (1955): Arch. Intern. Med., 95, 202.
3. Moore, D. C., Bridenbaugh, L. D., Green, J. C., Piccioni, V. S., Adams, P. A. and Lindstrom, C. A. (1955): J. Amer. Med. Assoc., 159, 1342.
4. Lebherz, R. B. and Harris, J. H. (1955): Obstet. and Gynec., 6, 606.

STELLENBOSSE BYDRAES

In hierdie uitgawe van die *Tydskrif* plaas ons die wetenskaplike bydraes—in Engels en in Afrikaans—wat gelewer is tydens die tweede akademiese jaardag van die Mediese Skool van die Universiteit van Stellenbosch en die Karl Bremer-Hospitaal te Bellville, op 5 September 1958. Hiermee word die gebruik dan ook vasgelê om gereeld spesiale ruimte af te staan in die orgaan van die Mediese Vereniging van Suid-Afrika vir bydraes van hierdie aard.

Dit wil vir ons voorkom of die presedent wat deur die publikasie van bydraes, gelewer by spesiale akademiese geleenthede, 'n goeie belofte vir die toekoms inhou. Mediese navorsingswerk aan ons verskillende universiteite en mediese skole word natuurlik gereeld gedoen en kan nie aan datums en tye vasgekoppel word nie. Nogtans sou dit goed wees as die tradisie in ons land sou kon ontwikkel om elke jaar tydens een of ander akademiese herdenkings- of feestelike geleentheid 'n keur van bydraes van al ons universiteite beskikbaar te maak vir publikasie in spesiale uitgawes van die *Tydskrif*.

THE RELATIONSHIP BETWEEN THE ALPHA AND BETA-CELLS IN THE ISLETS OF LANGERHANS OF THE ALBINO RAT *

MORPHOLOGY AND CYTOGENESIS

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'In spite of the voluminous literature on the morphology of the pancreas, there is as yet no agreement on such a fundamental point as the recognition of cell types in the islets of Langerhans' (Thomas[27] 1937). During the past 21 years a considerable additional number of papers dealing with this subject have been published; the problem, however, remains largely unsolved.

Thus, according to Thomas, 'A, B and D cells have been identified by their differential coloration and constant nuclear qualities in the islets of all forty-one species of mammals examined' (including 10 species of rodents), whereas Jewell and Charipper[17] could find no D-cells in the islets of the golden hamster. Gomori[8] stated that 'there is no accepted routine procedure by which the cell types in the pancreatic islets can be identified with invariable certainty' and proposed a new modification of the Mallory-Heidenhain-azan stain suitable for the study of the islet cells. This was followed (Gomori[9]) by an improvement of the chromium-haematoxylin phloxin method with which, according to Gomori, 'D-cells are indistinguishable from alphas.'

This lack of agreement on the recognition of cell types in the islets of Langerhans is overshadowed by the existing controversy on the *origin* of the cell types of the islets and the possible *transitions* between the different cell types found in the pancreas as a whole. The present investigation was undertaken in the hope that it would throw some light on these controversies.

MATERIAL AND METHODS

The animals used in this study were of the Wistar strain *Rattus norvegicus* bred in the Department of Physiology, University of Stellenbosch. For studying the different cell types in the islets of the albino rat, female animals weighing between 170-200 g. were used, except for 6 animals which were very young and weighed between 15-19 g. Some of these animals received injections of alloxan while others were

* Paper presented at 2nd anniversary of the Stellenbosch Medical School, Bellville, 5 September 1958.

made diabetic by repeated injections of dextrose solution over a period of 21 days.

Regarding the study of the histogenesis and cytogenesis of the islets, embryos were collected at 6 different stages of development. Females in oestrus were placed with male animals at 10 a.m. and separated into individual cages at 2 p.m. The age of the embryos were arbitrarily computed from 12 noon.

Animals were killed by a sudden blow on the head and the pancreas or embryos quickly removed. The embryos were immediately decapitated and placed in the fixing solution. All materials were fixed in Bouin-solution for 10-12 hours, washed in running water for 8 hours and imbedded in paraffin wax. The adult material was sectioned serially at 3μ and stained according to Gomori's modification of the Mallory-Heidenhain-azan stain. The embryos were sectioned serially at 5μ and stained according to Gomori's improvement of the chromium-haematoxylin-phloxin method.

RESULTS

Experiment I. An Investigation of the Different Cell Types found in the Islets of Langerhans of the Albino Rat.

In this experiment 30 rats were used. They were divided into 3 groups: Group A was the normal control group. The animals of group B were injected with a freshly prepared 3% solution of alloxan in distilled water. The animals of group C were injected with a 30% solution of chemically pure dextrose. The protocols of these animals are listed in Table I.

TABLE I. PROTOCOLS OF THE 30 RATS

Group	*Rat No.*	*Body Weight (g.)*	*Injection and Dose*	*No. of Islet Sections Investigated*	*State of Islets*	*Cell Types*
A	I	180	—	200	Normal	A & B
	II	176	—	200	,,	A & B
	III	170	—	200	,,	A & B
	IV	16	—	200	,,	A & B
	V	17	—	200	,,	A & B
	VI	170	—	18,206	,,	A & B
	VII	175	—	19,107	Some degenerated	A, B & D
	VIII	180	—	17,983	Normal	A & B
B	IX	175	Alloxan-	200	Some degenerated	A, B & D
	X	180	20 mg./kg.	200	,,	A, B & D
	XI	190	body weight	200	,,	A, B & D
	XII	185	3X daily	200	,,	A, B & D
	XIII	175	for 21 days	21,288	,,	A, B & D
	XIV	180	,,	25,180	,,	A, B & D
	XV	180	,,	21,906	,,	A, B & D
	XVI	170	,,	22,679	,,	A, B & D
	XVII	175	,,	21,469	,,	A, B & D
	XVIII	180	,,	24,302	,,	A, B & D
C	XIX	201	Dextrose-	200	Some degenerated	A, B & D
	XX	187	0·75 g./kg.	200	,,	A, B & D
	XXI	19	body weight	200	,,	A, B & D
	XXII	18	3X daily	200	,,	A, B & D
	XXIII	15	for 21 days	200	,,	A, B & D
	XXIV	18	,,	200	,,	A, B & D
	XXV	180	,,	23,741	,,	A, B & D
	XXVI	180	,,	22,730	,,	A, B & D
	XXVII	175	,,	22,139	,,	A, B & D
	XXVIII	190	,,	25,652	,,	A, B & D
	XXIX	175	,,	23,469	,,	A, B & D
	XXX	180	,,	22,804	,,	A, B & D

Two distinct cell types could be distinguished in the normal islets with the aid of Gomori's method (Fig. 1), except in rat No. VII (Table I) where a third type (which will be referred to as type D) was found. Although rat No. VII was included in this series as normal, some of the islets of Langerhans showed an advanced state of degeneration which was, however, only detected after the total number of islets in the pancreas was investigated (Fig. 2).

In the normal islet-section the central part is occupied by cells of irregular form with an average cross-section of 14 μ.

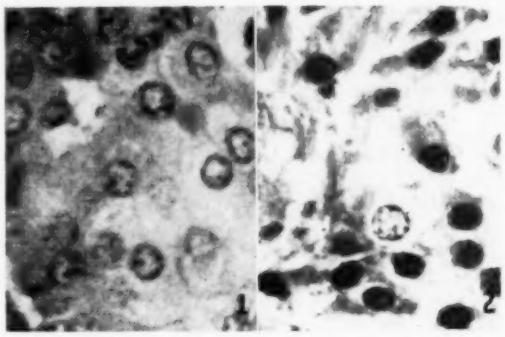

Fig. 1. Islet of Langerhans of normal rat. β-cells occupy the central part of the islet, while α-cells can be seen in the upper left-hand corner. X970. *Fig.* 2. Islet of rat No. VII (Table I). One 'D-cell' is shown among a number of degenerated β-cells with pyknotic nuclei. X970.

The cytoplasm stains a light orange-grey and is packed with fine dustlike cytoplasmic granules. These are considered to be β-cells. The second type is invariably found on the periphery of the islets, sometimes forming a complete ring encircling the β-cells, but often clumped together to form isolated groups on the periphery. They are on the average smaller than the β-cells, the cytoplasm stains somewhat more darkly and their cytoplasmic granules are more distinct with a faint red colour. These are considered to be α-cells. No difference in the structure of the nuclei of the α- and β-cells could be observed.

The 'D-cells' are characterized by being larger than both the α- and β-cells with an average cross-section of 18 μ. The nuclei are also larger with apparently less chromatin-substance, which is usually arranged in irregular clumps against the nuclear membrane. In some of these cells the nuclear membrane itself is damaged and karyorrhexis has taken place. The cytoplasm appears to be very faintly stained and not filled with granules. Some cells of this type were found with large portions of the cytoplasm totally devoid of granules. However, where present, these granules could not be distinguished from those of the normal β-cells. They were therefore considered to be β-cells which had undergone hydropic degeneration.

In some islets of the animals in groups B and C, as well as in some of rat No. VII (group A), a considerable number of cells with pyknotic nuclei was found. These cells were smaller than the normal α- or β-cells and were considered to be β-cells which had undergone a marked degree of *pyknotic degeneration*, in contradistinction to the *hydropic degeneration* of the 'D-cells'.

From these observations it must be quite clear that the *normal* islets of Langerhans of the albino rat are constituted of *two cell types* only, while in those made diabetic, either by injection of alloxan or by a persistent elevation of the blood sugar by repeated intraperitoneal injections of dextrose, a third cell type ('D-cells') is invariably encountered apart from the fully degenerated β-cells showing pyknotic nuclei (Figs. 3 and 4).

Experiment 2. A Study of the Origin and Cytogenesis of the Islets of Langerhans

Normal healthy female animals were paired with males and killed at different times after successful copulation had

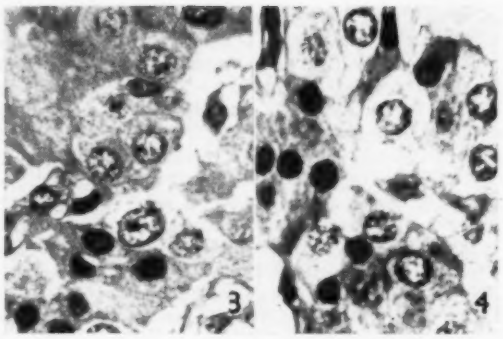

Fig. 3. Islet of alloxan-diabetic rat. Normal β-cells, 'D-cells' and degenerated β-cells with pyknotic nuclei can be distinguished. X970. *Fig.* 4. Islet of rat made diabetic by repeated injections of dextrose-solution; cells similar to Fig. 3. X970.

TABLE II. FINDINGS IN RAT EMBRYOS KILLED AT DIFFERENT TIMES

Rat No.	*Killed after*	*No. of Islets*	*Total No. of Islet-cross-sections*	*Average Islet-Vol. c.mm.* $\times 10^{-3}$	*Total Islet Vol c.mm.* $\times 10^{-3}$
XXXI	10½ days	None	—	—	—
XXXII	14½ days	"	—	—	—
XXXIII	17½ days	Islet-anlagen	—	—	—
XXXIV	19½ days	178	3,557	0·52	93·0
XXXV	21 days	233	4,787	0·57	132·3
XXXVI	22½ days	344	8,779	1·09	374·6

taken place (Table II). The embryos (one from each age-group) were serially sectioned at 5 μ and all the sections, after having been stained with Gomori's modification of the chromium-haematoxylin-phloxin method, were microscopically examined with the aid of the oil-immersion objective. The total number of islets as well as the total number of cross-sections through these islets were counted. From this the average volume of the islets was calculated from the formula $4/3\pi r^3$. The diameter (2·r) was obtained by dividing the number of islet cross-sections by the number of islets (assuming that the islets are all spherical) and multiplying by 5 μ i.e. the thickness of the sections. The following observations were made in this experiment:

At 10½ *days.* At this stage the ventral and dorsal pancreatic anlagen have developed and grown to such an extent that they have come in contact with each other, although complete fusion has not yet occurred. The cells are arranged in cords, some of which show distinct lumina (tubules) while others are nothing but solid masses of undifferentiated cells. Mitotic figures are very conspicuous, especially in the cells bordering the lumina of the tubules or those situated centrally in the solid cell cords. At this stage no structures are present that can properly be designated *islets of Langerhans.*

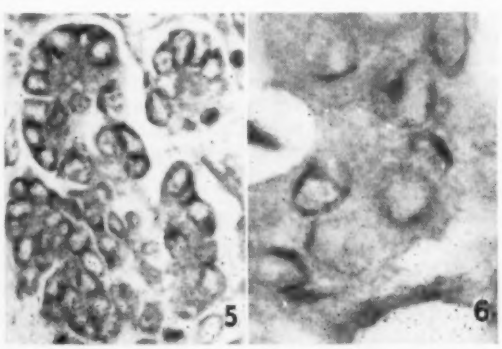

Fig. 5. Portion of pancreas of 17½-day embryo. An acinus-like structure is seen with centrally-situated cells similar to the centro-acinar cells of the adult pancreas. X450. *Fig.* 6. 'Primitive islet' from 17½-day embryo. It is seen in close relation to a pancreatic tubule. X970.

At 17½ *days.* The pancreas at this stage consists chiefly of tubules and solid cell cords embedded in embryonic connective tissue. Occasionally a tubule (now a secretory duct) ends in an acinus-like structure with one or more centrally-situated cells similar to the centro-acinar cells of the adult pancreas (Fig. 5). Mitotic figures are often present in these cells. Dispersed among these structures are found a few irregular masses of cells that are considered anlagen for future islets of Langerhans. Fig. 6 shows one of these *primitive islets* in close relation to a pancreatic tubule. Some of the cells can best be described as medium-sized with irregular shape and finely granular cytoplasm resembling that of the β-cells of adult islets of Langerhans.

Apart from these 'islets' a few cell masses are present that could easily be erroneously designated *primitive islets.* They are, however, nothing but conglomerates of connective tissue cells, as judged by their cytoplasmic processes. As seen from Table II the 'islets' were not counted because they were considered in no wise comparable to adult islets of Langerhans.

At 19½ *days.* Some of the 'islets' at this stage have developed to such a degree that they can be enumerated as distinct units. They should, however, not be considered 'islets of Langerhans' because they do not yet possess the two types characteristic of the mature islets—alpha cells are still absent.

At 21 *days.* More 'islets' are present, and the larger ones are more numerous (17% greater than 150 μ in cross-section as compared to 12% at 19½ days). The average cross-section of the islets is, however, not appreciably greater than at 19½ days. This is due to the presence of a larger percentage of small, newly-formed 'islets'.

A very interesting phenomenon was noticed at this stage: several cells, showing beta granules, were found embedded among the cells of the pancreatic ducts, especially the intercalated ducts (Fig. 7). These are considered to be β-cells which differentiate directly from the duct-epithelium, in contrast to the β-cells previously encountered at the 17½ and 19½-days stage. These latter were invariably present among massas of undifferentiated cells (the primitive islets) and are considered to develop from these.

At 22½ *days.* Thirty-one per cent of the islets have now reached a size of 150 μ or more in cross-section, and the total number of 'islets' has nearly doubled since the 19½-days stage. The total 'islet-volume' has more than doubled during this period. This enlargement of the islets must be attributed to mitotic activity within the islets—not only were mitoses observed in the undifferentiated cells of the primitive islets but mitotic figures are also found in the well-differentiated β-cells. The increase in the total 'islet-volume' is effected by enlargement of the existing islets as well as the development of new islets from the anlagen.

At this stage a *new cell type* also differentiates from the tubule-epithelium. Fig. 8 shows a cell of this type with characteristic alpha granules arranged in such a way as to

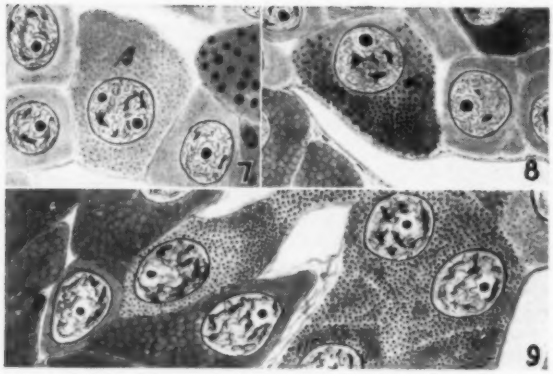

Fig. 7. Section through pancreatic tubule from 21-day embryo. A fully differentiated β-cell is shown. *Fig.* 8. Section through pancreatic tubule from 22¼-day embryo. An α-cell with distinct polarity towards the capillary is shown.
Fig. 9. Section of pancreas taken from rat treated with $CoCl_2$. An acinus (left) adjacent to an islet (right) is shown. An α-cell, situated among the acinar cells and in contact with the centro-acinar cell, can be distinguished.
(*Figs.* 7-9 were drawn at a uniform magnification; oil immersion.)

give it a distinct polarity towards the capillary and away from the lumen of the intercalated duct.

Addendum

During the course of an investigation, under way, on the destruction of the alpha cells by injection of $CoCl_2$ it was noticed that new α-cells are regenerated by differentiation from the centro-acinar cells. Fig. 9 shows an alpha cell situated between the cells of an acinus and still in contact with a centro-acinar cell, presumably the sister cell of the one under investigation. The polarity of this cell is similar to that of the α-cell described at the 22¼-days stage.

DISCUSSION

The cell types in the islets of Langerhans. Laguesse[20] (1894) was the first to observe the existence of more than one cell type in the islets of Langerhans. Since then, different investigators paid attention to these different cell types and Lane[21] succeeded in identifying two kinds of granules in the islet cells which differed chemically from each other and from the zymogen of the acini. These are now generally known as alpha and beta granules (present in the α-cells and β-cells, respectively). In addition to the α- and β-cells three other cell types were distinguished by different authors in different animal species: Bensley[1] described cells in the islets of the guinea pig and called them C-cells (gamma cells; Gomori[8]) and Bloom[2] (1931) described the so-called D-cells in the human islets of Langerhans. D-cells were found in 41 species of mammals (including the albino rat) by Thomas,[27] who also identified a fifth type of cell (E-cells) in the opossum islets.

With the staining methods of Gomori[8, 9] it was invariably possible to distinguish *two cell types* in the normal islets of the rat, except in some of the smaller islets in which only β-cells could be found. The differentiation of at least two cell types in adult pancreatic islets is further supported by their differential phosphatase activity (Gomori,[10] Jacoby[15] and McAlpine[22]) and the electron-microscopic investigation by Lacy.[19]

Concerning the C-cells conflicting evidence appears to exist: Cowdry[4] stated that 'A, B and C cells can be accepted without reservation as fundamental components of the islets'. According to Gomori,[8] however, gamma cells (C-cells) are merely beta cells which are very poor in, or possibly devoid of specific granules. Bremer and Weatherford[3] stated that 'the C-cell may represent a younger form of the A-cell'. E-cells were described in the islets of the opossum only. The D-cells, in my opinion, are nothing but cells in a certain phase of activity or degeneration, comparable to the β-cells undergoing hydropic degeneration. This opinion is in partial agreement with the statement of Gomori[9] that 'D cells are probably aged alpha cells'.

The origin of the islets of Langerhans. The embryonic islets differentiate from the entoderm and do not show any genetic relationship to the mesodermal tissue in which they become embedded. The first 'islet-anlage' could be distinguished in the 17¼-day embryo. No 'islets' were encountered in the 14¼-day embryo, a finding which conflicted with Hard's statement[12] that the first 'islets' differentiated in the 13-day embryo. It must, however, be stressed that in the present investigation only those structures with a marked cellular compactness comparable to the adult islets of Langerhans, were counted as 'islets'.

From 17¼ days onward 'islets' differentiated and increased in size at such a rate that at 22¼ days the total volume reached the value of 374×10^{-3} c. mm. (Table II). This volume, however, constituted only a very small part of the islet-tissue of the adult as estimated by Haist and Pugh.[11] One might therefore conclude that only a small percentage of the future islets of Langerhans originate during intra-uterine development.

The cytogenesis of the β-cells. Gomori[9] stated that 'the origin of beta cells is unknown'. Hard[12] was able to distinguish granules in the islet cells at the 19-day stage of embryonic development, while McAlpine[22] described 'a new cell type' at 18 days and identified this type as the beta cell. In my material specific granules were first seen at 17¼ days, which is in good agreement with the findings of McAlpine.

It was concluded that these first recognizable β-cells originated from undifferentiated cells which budded from the pancreatic ducts. Later on β-cells differentiated directly from the duct-epithelium, while some cells originated as a result of mitosis of existing β-cells. These results suggested a threefold origin of the β-cells. This conclusion conflicted, however, with that of Ferner and Stoeckenius[6] who also claimed that three possibilities existed concerning the cytogenesis of the β-cells, but according to whom β-cells might originate *inter alia* by the transformation of α-cells into β-cells.

The cytogenesis of the α-cells. According to Hard[12] 'the beta cell is the only islet cell type to differentiate during embryonic development', and 'alpha cells are first recognized during the second day of postnatal life'. This opinion was not substantiated by the present study because occasional α-cells (with specific granules) were distinguished at 22¼ days. Although Hultquist and Thorell[14] believed that 'α-cells . . . differ with respect to their ultraviolet cytology in budding germs and in islands of the adult type', I was unable to make this distinction.

In contrast to the suggested threefold origin of the β-cells, the α-cells showed a monophyletic origin *viz.* differentiation

from the duct-epithelium. Mitotic figures were never encountered in the α-cells of my material. This is supported by Jaffe,[16] but conflicts with the finding of Mosca[23] who could see mitotic figures in both the α- and β-cells. Even after $CoCl_2$-treatment the proliferation of α-cells in the adult occurred from duct-epithelium (the centro-acinar cells).

The relationship between the different cell types of the pancreas. A number of authors (Dale,[5] Woerner,[30] and Johnson[18]) described transition-cells which showed characteristics of both islet- and acinar cells. From this they suggested that acinar cells could be transformed into islet-cells. Vincent,[29] Otani[24] and Sergeyeva[25] claimed that transformation of acinar cells into islet-cells and *vice-versa* occurred even in adults. On the other hand Tschassownikow,[28] working on the axolotl, stated that even the *possibility* of islets being transformed into glandular acini was excluded in the light of his observations. He therefore did not accept the 'theorie de balancement Laguesse'.

In the present study no transition-cells which showed characteristics of both acinar and islet-cells were found; it was concluded, therefore, that the islets of Langerhans were morphological entities *sui generis*. This conclusion is supported by Freise,[7] Hirata[13] and Gomori.[9]

Ferner and Stoeckenius' statement[6] that α-cells can be transformed into β-cells is mistaken in my opinion. It is also in marked contrast to the findings of Terbrüggen,[26] Gomori[9] and others. If the α-cells were to be taken as the precursors of the β-cells there is no possible explanation for the fact that the β-cells invariably differentiated during the embryonic development several days before there was any sign of specific α-granules (and thus of α-cells). Also, it will be very difficult to explain the observation that destruction of the β-cells with alloxan (Esterhuizen, 194[illegible]—unpublished results) is permanent, whereas destruction of the α-cells with $CoCl_2$ is followed by marked regeneration.

SUMMARY

1. The cellular composition of the islets of Langerhans in normal and diabetic rats was investigated. Rats were made diabetic by (*a*) injections of alloxan and (*b*) repeated injections of dextrose solution. The normal islets of Langerhans of the albino rat are constituted of two cell types, while in those made diabetic a third type ('D-cells') was invariably encountered apart from the fully degenerated β-cells showing pyknotic nuclei.

2. Regarding the origin and cytogenesis of the islets it was found that the first 'islet-anlage', as well as specific beta-granules, could be distinguished in the 17½-day embryo. Alpha-cells were first distinguished at 22½ days. It is suggested that the β-cells have a threefold and the α-cells a monophyletic origin.

3. No transition-cells which showed characteristics of both acinar and islet-cells were found. It is concluded, therefore, that the islets of Langerhans are morphological entities *sui generis*.

I wish to thank Prof. H. E. Brink for his interest in this work and Dr. M. W. P. Nienaber for his help in preparing the manuscript.

All photomicrographs were taken through a Wratten K_1 filter.

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NAALDBIOPSIE VAN DIE PLEURA*†

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Pleurale effusies kan tergende diagnostiese probleme lewer en aangesien die toestand beskou moet word as 'n potensieelernstige toestand, is vroeë diagnose altyd gewens. Dit is egter juis in hierdie toestand waar 'n etiologiese diagnose dikwels eers laat gemaak word. Die doel van hierdie publikasie is om die metode van naaldbiopsie van die pariëtale pleura as 'n nuttige prosedure vir die vasstelling van 'n etiologiese diagnose in gevalle met idiopatiese pleurale effusie aan te beveel, en die aandag van veral die algemene praktisyn te vestig op die algemeen-toepasbare moontlikhede van hierdie tegniek.

TEGNIEK

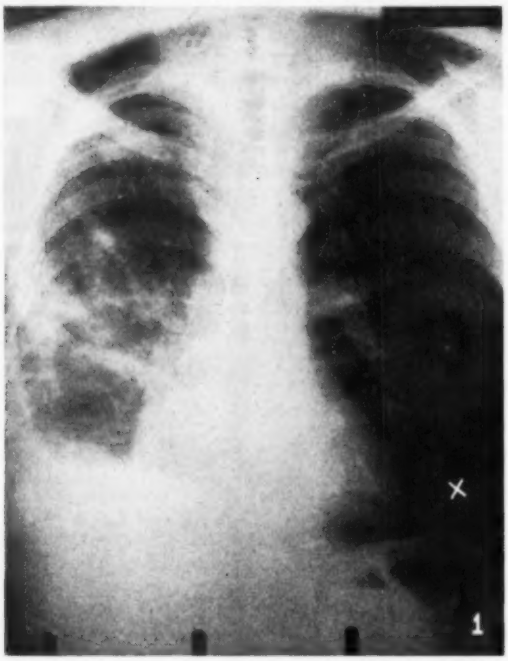
Die tegniek wat ons gebruik is beskryf deur de Francis, Klosk en Albano[1] met die volgende wysigings: (1) 'n Velsnit word nie gemaak nie, maar die ingangspoort van die aspirasienaald word gebruik vir die Vim-Silvermannaald. (2) Arterieklemme word gebruik in plaas van Kellyklemme, omdat ons dit geriefliker vind vir persoonlike gebruik en ons meen dat 'n arterieklem, vanweë sy groter lengte en gewig, die biopsienaald stabiliseer.

Voordat tot biopsie oorgegaan word, word die vog eers geaspireer vir marmotinspuiting, en direkte ondersoek na tuberkel basille en 'n monster vir sitologiese ondersoek word

* 'n Reeks van die resultate in 20 gevalle is as uitstalling aangebied by die Karl Bremer-Hospitaal se tweede akademiese jaardag op 5 September 1958.

† 'n Oorsig van die vraagstukke in verband met pleurale biopsies is op 19 Julie 1958 in die *Tydskrif* bespreek in 'n redaksionele artikel (**32**, 719).

ook geneem (na biopsie mag daar bloed by die vog wees). Hierna word die effusie volledig geaspireer en opvolg-roentgenfoto's word geneem. Laasgenoemde prosedure dien 'n dubbele doel: (1) Onderliggende patologie mag nou sigbaar word, bv. 'n tuberkuleuse infiltraat (Afb. 1) en (2) 'n Area van maksimale verandering in die long of pleurale verdikking



Afb. 1. Roentgenfoto van borskas. Massiewe regssydige pleurale effusie na aspirasie. Toon 'n infiltraat in regter-longapeks en middelkwab; verdag van tuberkulose.

mag sigbaar word en, indien die eerste biopsie nie spesifiek was nie, dui hierdie veranderings die mees geskikte plek vir die volgende biopsie aan.

In al die gevalle word 'n roetine Mantoux-toets gedoen. 'n Negatiewe Mantoux-toets spreek sterk teen die moontlikheid van tuberkulose as etiologiese faktor en behoort die vasstelling van 'n histologiese diagnose gebiedend noodsaaklik te maak. Roper en Waring[2] het in 141 gevalle met serofibrineuse pleurale effusie by almal 'n positiewe Mantoux-reaksie gevind en van diegene wat onder 25 jaar oud was, het 75·3% aktiewe tuberkulose ontwikkel (onbehandel) binne 5 jaar. Diegene ouer as 25 jaar het waarskynlik, indien onbehandel, 'n beter prognose aangesien slegs 54·4% van hulle aktiewe tuberkulose gedurende die tyd van studie ontwikkel het. Almal in ons reeks wat 'n histologiese bewys van tuberkulose getoon het, het 'n positiewe Mantoux-reaksie gehad, en by diegene met 'n negatiewe reaksie, maar by wie die siekte klinies, soos by die ander, met 'n sogenaamde ‚idiopatiese pleurale effusie' begin het, is ander oorsake vir die effusie gevind.

RESULTATE

Soos Donohue *et al.*,[3] het ons ook 'n groep gevind met niespesifieke inflammatoriese veranderings in die pleura wat volgens kliniese indruk 'n sneller genesing en geringer kliniese simptomatologie toon as die tuberkuleuse groep. Hierdie groep word nog opgevolg en meer gekontroleerde studies is nodig alvorens ‚benigne pleuritis' as kliniese entiteit, verwant aan benigne perikarditis, aanvaar kan word — soos tereg voorgestel deur die genoemde skrywers. Vir praktiese doeleindes moet 'n idiopatiese pleurale effusie as tuberkuleus of maligne beskou word tot tyd-en-wyl histologiese bewys van 'n ander oorsaak gevind word. Ons resultate word aangegee in Tabel I.

BESPREKING

Die meeste mislukkings om pleura te verkry met hierdie tegniek het, soos by al die vorige reekse, vroeg in ons ondervinding voorgekom. Na 6 biopsies was daar 3 mislukkings, en na die 15e biopsie het dit ons weer misluk om pleura te verkry. In 3 gevalle is pleura eers by die tweede poging verkry.

Voorbeelde van die histologiese bevindings word in Afbs. 2-5 gegee. Afb. 5 toon aan die grootte van die monster pleura. Hierdie is 'n besonder klein monster; gewoonlik is dit 2-3 maal groter. Dikwels is dit nie makroskopies moontlik om die weefsel as pleura of spier te identifiseer nie en alle brokkies wat tussen die blaaie van die Vim-Silvermannaald se biopsie-skag gevind word, moet vir histologiese ondersoek gestuur word.

In die gevalle met 'n tuberkuleuse effusie is, benewens 'n positiewe Mantoux-reaksie, slegs in een geval 'n positiewe resultaat na marmotinspuiting verkry, en in een geval (Afb. 1) het radiologiese ondersoek ook die diagnose bevestig na volkome aspirasie. Sputa en nugter-maagsapondersoeke was in al die gevalle negatief.

In die geval met mesotelioom van die pleura (Afb. 3) het ons 'n paar punte gevind wat verdere bespreking regverdig: Die pasiënt was 'n jong blanke vrou, 32 jaar oud. Sy was volkome gesond tot 3 weke voor opname, toe sy na 'n griep-aanval toenemende dispnee ondervind het, later ook met rus. Die enigste fisiese bevinding was dié van 'n massiewe linkssydige pleurale effusie in 'n baie dispneuse vrou. Die Mantoux-reaksie was negatief en 'n bloederige effusie is geaspireer. Die biopsie toon 'n tumor met 'n asinêre struktuur soos 'n adeno-karsinoom, hoewel soortgelyke beelde in mesotelioom gevind kan word. Die pleurale vog toon by sitologiese ondersoek selle verdag van maligniteit.

Die tiroïed, ginekologiese organe en mammæ is weereens sorgvuldig nagegaan om 'n primêre tumor uit te skakel, en radiologiese ondersoek van die spysverteringskanaal en niere is gedoen, terwyl bronchoskopie ook insgelyks negatief was.

Die torakale roentgenondersoeke het nou, behalwe verdikte pleura, 'n verbetering in die beeld getoon in soverre die effusie nie weer opgevul het nie. Operasie het 'n verdikte pleura getoon, op snit was daar die stewigheid van kraakbeen in areas en vriessnitte het dieselfde histologiese beeld getoon as by naaldbiopsie. Die pleura het maklik en volledig gestroop.

'n Mens sou dus geneig wees om, sonder die histologiese diagnose, 'n afwagtende houding in te neem. Vroeë diagnose in hierdie geval mag dus die prognose, aangegee as swak vanweë laat diagnose,[4] gunstig beïnvloed.

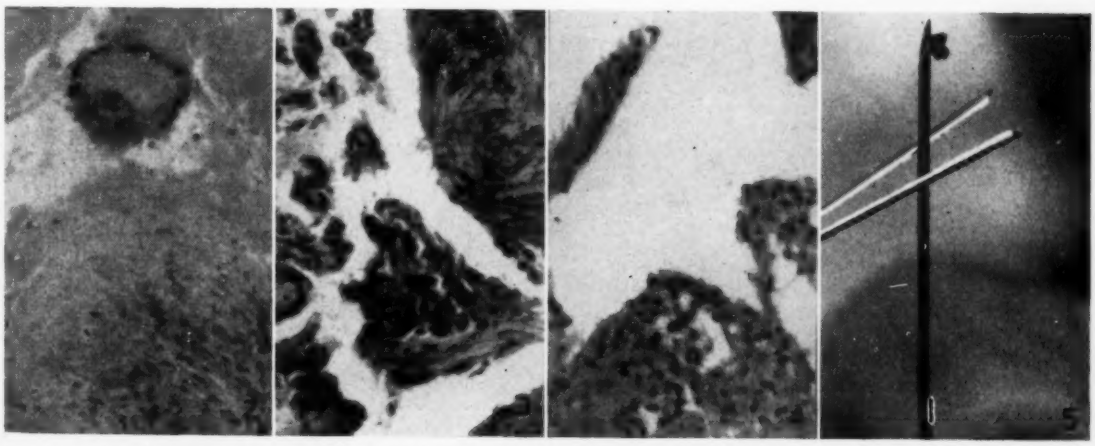
In Tabel II word die gevare, komplikasies, morbiditeit en mortaliteit by naaldbiopsie aangegee terwyl Tabel III 'n weergawe is van die respektiewelike voor- en nadele van die chirurgiese en naaldbiopsie van die pleura soos in die literatuuroorsig van Donohue *et al.*[3] gemeld, en soos ons dit self insien. In hierdie reeks was daar geen mortaliteit nie en die enigste komplikasie was 'n klein pneumotoraks wat slegs

TABEL I. OPSOMMING VAN RESULTATE

Biopsie spesifiek	*Tuberkulose*	*Magliniteit*	*Biopsie nie-spesifiek*	*N.S.P.*	*Chron. inflammatories*	*Akuut inflammatories*
6	5	1	10	3	6	1
Mantoux 1/1000: 5 positief 1 negatief	Positief al 5 gevalle	Negatief	5 positief 3 onseker 2 negatief	1 positief 1 onseker 1 negatief	3 positief 2 onseker 1 negatief	Positief
Aard van vog: 5 strooikleurig 1 bloederig	Strooikleurig	Bloederig	8 strooikleurig 1 waterhelder 1 etter	Strooikleurig	5 strooikleurig 1 waterhelder	Etter
Sitologie: 5 negatief 1 positief	5 negatief	Positief	3 limfosiete 7 nie-spesifiek	1 limfosiete	2 limfosiete	Anaerobiese Streptokokkus
Marmot-inspuiting: 1 positief 5 negatief	Positief in 1 geval. (Neem 6 weke)	Negatief	Negatief	Negatief	Negatief	Nie gedoen
Tydsduur: 1. Die histologiese verslag beskikbaar 48-72 uur na biopsie. 2. Marmotinspuitings neem 6 weke of meer.			*Roentgenfotos:* Van geen spesifieke diagnostiese hulp nie, behalwe lokalisasie van die effusie, ens.			
Roentgenfotos: Slegs in 1 geval diagnosties	Toon Koch-infiltraat in 1 geval	Verdikte pleura maar geen spesifieke hulp nie	*Geen pleura nie:* 4- 2 met hartversaking en massiewe effusie. 1 prostaatmetastases. 1 longtuberkulose, amiloidose en nefrose.			

N.S.P.= Nie-spesifieke pleuritis. Chron. inflammatories= Sluit in gevalle wat anti-T.B.-behandeling ontvang het. Ak. inflammatories= Gekapselde empieem.

Aantal gevalle: 20. Blankes: 9 (5 mans, 4 vrouens). Nie-blankes: 11 (7 mans, 4 vrouens).



Afb. 2. Naaldbiopsie pleura. Toon area met homogene nekrose en verkasing omring deur epiteloïede selle.

Afb. 3. Naaldbiopsie pleura. Toon maligne tumor met klierstruktuur wat histologies die beeld van adenokarsinoom toon maar ook gevind kan word in mesoteliomata.

Afb. 4. Naaldbiopsie pleura. Nie-spesifiek-inflammatoriese veranderings. Diffuse infiltraat met limfositiese selle.

Afb. 5. Naaldbiopsie van die pleura. Toon die grootte van die monster. Hierdie monster is besonder klein. Gewoonlik word 'n monster 2 tot 3 maal hierdie grootte verkry.

TABEL II. KOMPLIKASIES, ENS. BY NAALDBIOPSIE

Gevare	*Komplikasies*	*Morbiditeit*	*Mortaliteit*
1. Pleurale skok	1. Empieem	Soos vir pleurale aspirasie	Geen in meer as 100 gerapporteerde gevalle.[3]
2. Trauma aan (*a*) Interkostale bloedvate (*b*) Interkostale senuwees (*c*) Long	2. Hemotoraks 3. Spanningspneumotoraks		

Ons enigste komplikasie was geringe pneumotoraks radiologies gevind.

TABEL III. DIE VOOR- EN NADELE VAN CHIRURGIESE EN NAALDBIOPSIEMETODES

	Voordele	*Nadele*
Chirurgies	1. Meer akkuraat as volledige torakotomie ook gedoen word. 2. Behandeling indien chirurgies kan direk toegepas word, bv. maligniteit in vriessnit.	1. Vereis chirurg, narkotiseur en teater. 2. Hoër onkoste. 3. Nie toepaslik in alle gevalle vroeg in siekte nie. 4. Komplikasies meer, bv. empieem. embolisme en mortaliteit hoër. 5. Morbiditeit en mortaliteit hoër.
Naaldbiopsie	1. Net so akkuraat soos chirurgiese biopsie sonder volledige eksplorasie. 2. Eenvoudig, herhaalbaar en vroeg in siekte toepaslik. 3. Geringe morbiditeit. Dusver geen mortaliteit rapporteer. 4. By opname gedoen, is histologie na 48-72 uur beskikbaar. 5. Vereis slegs een geneesheer.	1. Geen—akkuraatheid word verhoog deur die herhaalbaarheid van die prosedure.

radiologies gevind is en wat na gewone aspirasie ook mag voorkom. Die morbiditeit vind ons dieselfde as vir 'n gewone pleurale aspirasie.

OPSOMMING

1. Ons ondervinding met naaldbiopsie in 20 gevalle met pleurale effusie word bespreek.

2. Die metode word aangewys as 'n nuttige, eenvoudige en veilige prosedure.

3. Die belang van die prosedure in die vroeë diagnose van maligne pleurale letsels word geïllustreer met beskrywing van 'n geval met 'n mesotelioom van die pleura.

4. Mikrofotos van die histologiese bevindings by 'n paar gevalle word afgebeeld.

SUMMARY

1. Our experience with needle biopsy of the parietal pleura, using the technique as described by de Francis, Klosk and Albano,[1] with two slight modifications, is discussed.

2. This method is recommended to all practitioners, who aspirate pleural effusions, as a safe, simple and valuable procedure.

3. The importance of this procedure in establishing an early diagnosis in malignant pleural lesions, is illustrated by the description of a case with a mesothelioma of the pleura.

4. Microphotographs of the histological picture obtained in some of the cases are depicted.

ADDENDUM

Sedert die ter perse gaan van hierdie artikel is nog 5 verdere gevalle van maligniteit op hierdie wyse ontdek: 3 gevalle met metastatiese karsinoom (prostaat 2, maag 1), wat as pleurale effusies van onbekende etiologie gepresenteer het; 1 geval met limfosarkoom (14-jarige Kleurlingvrou) en 1 geval van waarskynlik primêre pleurale maligniteit, hoewel 'n perifêre karsinoom van die bronchus nie met sekerheid uitgeskakel kon word nie.

Samuels *et al.*[5] beskryf 'n atipiese fibrose-beeld wat maligniteit suggereer en hulle voel dat die korrekte beskrywende term ‚reactive fibrosis of tumour-induced type' sal wees. Een geval uit ons groep ‚chronies inflammatories' sal hierby inpas en die verdere verloop van die geval suggereer maligniteit van die pleura.

Ek wil graag my dank uitspreek aan prof. A. J. Brink vir fasiliteite en advies in verband met die werk, aan prof. H. Weber vir die histologiese en sitologiese ondersoeke, aan mnr. R. Ellis vir die fotografie, aan mej. Y. Stuart vir die tikwerk en aan dr. R. L. M. Kotze, Superintendent, Karl Bremer-Hospitaal vir toestemming tot publikasie.

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FIBROSIS IN RATS' LUNGS PRODUCED BY RADIOACTIVE MINE DUST*

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Jones,[1] after having examined the mineral residues of 29 silicotic lungs, concluded that sericite (the hydrated silicate of aluminium and potassium) rather than free silica (quartz) was the main cause of silicosis. He also maintained that the gold-bearing quartz conglomerate (banket) of the Transvaal, South Africa, was one of the most notoriously dangerous rocks with respect to its silicotic effect. The gold-bearing quartz of the Kolar Gold Fields, India, although it contained more quartz than the South African rock, was found less pathogenic. Jones[1] ascribed the greater fibrotic activity of the

* Paper presented at 2nd anniversary of the Stellenbosch Medical School, Bellville, 5 September 1958.

South African rock to the presence of acicular fibres of sericite. These fibres were absent from the Kolar quartz or occurred very rarely.

Many authors (Fallon and Banting,[8] Lemon and Higgins,[10] Simson and Strachan,[11] and Cummins[7]) investigated the fibrotic activity of sericite with quite contradictory results. The general consensus of opinion, however, is that of the two, free silica is the more dangerous component (Mavrogordato[4]). No acceptable explanation has yet been offered for the extremely dangerous silicotic effect of the S.A. gold bearing rock and it occurred to us that the presence of radioactive material in the S.A. rock might be a possible contributing factor in the pathogenesis of silicotic fibrosis.

Animal experiments were therefore planned to examine the fibrotic response caused by radioactive and non-radioactive samples of mine dust.

MATERIAL

All the dust samples used in this experiment, except snowit, were modifications of an original dust sample obtained from one of the gold mines on the Witwatersrand, Transvaal, South Africa, which also produces uranium. The radioactivity of the samples was determined by means of a labgear model D4019/W scintillation counter with a D4019/L discriminator-unit set at 15 volts. The output pulses were counted with a Philips electronic counter. One-g. samples were used and the counting time was one minute. Silica determinations were also carried out according to the method described by Stacy and King.[5] The following samples were used:

1. R_{153}. Original mine dust with 153 output pulses per minute and a silica content of 84%.

2. R_{502}. This sample was prepared from the R_{153} dust by sedimentation in distilled water and subsequent centrifuging of the supernatant suspension. The very fine dust obtained by this procedure gave 502 pulses per minute and contained 66% of silica.

3. R_0. Non-radioactive dust was prepared from sample R_{153} by treating it with acid to remove its radioactivity. (Snell and Snell.[6]) The silica content of this R_0 dust was 86%.

4. R_{300}. This sample was prepared by mixing equal weights of R_{502} and R_0 and gave 300 pulses per minute. It contained 76% of silica.

5. Snowit (Belgian quartz). It contained 94% of silica by chemical analysis and showed no radioactivity.

All the fractions except R_{502} were ground in an electrically

TABLE I. SIZE DISTRIBUTION OF DUST PARTICLES AND SILICA CONTENT

	Snowit	R_{502}	R_{300}	R_{153}	R_0
$>10\mu$	1%	3·3%	2·1%	0·5%	0·8%
5-10μ	4%	18·8%	9·5%	1%	1·7%
$< 5\mu$	95%	77·9%	88·4%	98·5%	97·5%
% Silica	94	66	76	84	86

driven agate mortar for at least 24 hours. The particle-size distribution of the samples was determined microscopically (Table I).

Preparation of Dust Suspensions for Injection

Of each sample 2 g. were accurately weighed out into screw-capped bottles, 40 ml. of sterile physiological saline added to each and then sterilized in a boiling water bath for 30 minutes. In all the samples 1 ml. of dust suspension corresponded to 50 mg. of the individual dust, and each animal received this amount.

Animals. Five groups of 20 rats (*Rattus norvegicus*, Wistar Institute) were injected with one each of the 5 samples in doses of 50 mg. of dust per animal. The intratracheal-injection technique of King *et al.*[2] was used.

Duration of experiments. The experiments lasted 320 days and the rats were killed at regular intervals as indicated in Table II. Owing to a few deaths duplicate killings could not

TABLE II. DAYS OF SURVIVAL AND GRADE OF FIBROSIS

Days	*Snowit*	R_{502}	R_{300}	R_{153}	R_0
30	3	2	1	1	1
60	3	2	1	1	1
90	4	2	2	2	1
120	4	2	2	2	1
150	4	2	2	2	1
180	4	3	3	2	2
210	5	3	3	2	3
240	5	3	3	3	3
280	5	4	3	3	3
320	5	4	4	3	3

be performed in all cases. The lungs of the animals that died were discarded after post-mortem examination.

Histopathological technique. The lungs were fixed in 10% neutral formol saline and suitable blocks selected along their long axes at the level of the hilum. Serial sections were made at 6 μ and were stained with haematoxylin and eosin and by silver impregnation (Gordon and Sweet[3]). The sections were examined and the pulmonary fibrosis graded according to King *et al.*[12] Five grades of fibrosis were recognized: Grade I, loose reticulin fibres with no collagen; Grade II, compact reticulin with or without a little collagen; Grade III, somewhat cellular but made up mostly of collagen; Grade IV, wholly composed of collagen fibres and completely acellular; and Grade V, acellular, collagenous and confluent. The gradings and days of survival are summarized in Table II.

PATHOLOGICAL FINDINGS

Macroscopic appearance of the lungs. In comparing the gross appearances of the lungs, R_{153} and R_0 samples appeared to be the least harmful; the R_{300} and R_{502} fractions were more pathogenic and snowit the most pathogenic. Greyish irregular dust foci were visible subpleurally in all animals killed. The size of these lesions increased steadily and became firmer in consistency, especially in the Snowit, R_{502} and R_{300} groups. The hilar lymph nodes gradually increased in size, the increase being the greatest in the snowit group.

Microscopic study of the lungs. The pulmonary changes caused by the various dust modifications varied in rate and severity. In the snowit rats fibrosis developed more rapidly than in the other groups and by 30 days Grade III fibrosis was obtained. The lesions progressed fairly rapidly and by 210 days Grade V fibrosis was reached.

The various mine dust modifications produced more or less the classical lung pathology of experimental silicosis, but fibrosis developed more slowly than in the snowit group. The R_{502} and R_{300} samples were more pathogenic than samples R_0 and R_{153}. They produced an advanced Grade III fibrosis by 180 days, while the R_0 and R_{153} groups only reached Grade II. Photomicrographs of the lesions are shown in Figs. 1-5.

From the sections examined it also appeared that relatively more cell destruction occurred in the lungs of the R_{502} and R_{300} groups than in the other groups especially during the early stages of the experiment. The fibrosis was more diffusely scattered throughout the lungs, and not mainly nodular in character as in the snowit group.

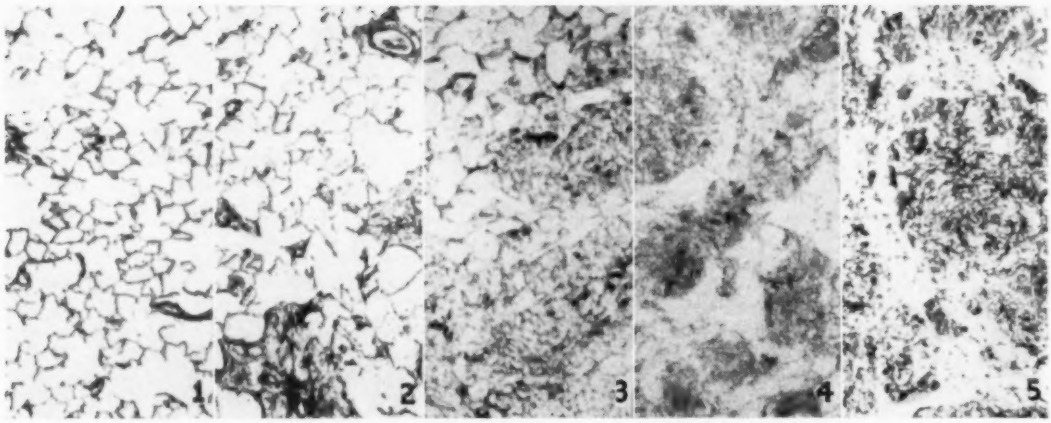

Fig. 1. Rat lung, 150 days after the injection of 50 mg. non-radioactive mine dust (R_0) with a silica content of 86% showing early Grade I fibrosis. Silver impregnation. (X 200). *Fig.* 2. Rat lung, 150 days after the injection of 50 mg. radioactive mine dust (R_{153}) with a silica content of 84% showing early Grade II fibrosis. Silver impregnation. (X 200). *Fig.* 3. Rat lung, 150 days after the injection of 50 mg. radioactive mine dust (R_{300}) with a silica content of 76% showing Grade II fibrosis. Silver impregnation. (X 200). *Fig.* 4. Rat lung, 150 days after the injection of 50 mg. radioactive mine dust (R_{502}) with a silica content of 66% showing an advanced Grade II fibrosis. Silver impregnation. (X 200). *Fig.* 5. Rat lung, 150 days after the injection of 50 mg. snowit with a silica content of 94% showing Grade IV fibrosis. Silver impregnation. (X 200).

Hyperplasia of the peribronchial lymphoid tissue was a common feature of all the sections examined, and in advanced fibrotic nodules scattered collections of lymphocytes were observed near the blood vessels and bronchial tubes.

Another prominent feature was the presence of needle-shaped, probably crystalline, structures in the lungs of the rats injected with R_{502} and R_{300}. In the R_0 and snowit groups these structures could not be detected while in the R_{153} group only a few 'crystals' were seen in some of the sections. These 'crystals' were especially abundant in the acellular nodules of the R_{502} and R_{300} series, but were also observed in some of the macrophages, in some alveoli and in a few loosely arranged cellular fibrotic nodules. There was a conspicuous new formation of canals in some of the advanced fibrotic areas caused by R_{502}, R_{300} and snowit samples.

DISCUSSION

In the present investigation snowit served as a control and it produced more fibrosis than the mine dust samples. No correlation could be established between the silica content of the different dust samples and the grade of fibrosis produced during the first 150 days of the experiment. Although sample R_{502} contained only 66% silica, it was more pathogenic than the original dust sample R_{153} which contained 84% silica. This finding suggests the presence of a possible fibrogenetic factor other than the silica content.

Needle-shaped structures were observed in the lung sections of the rats injected with samples R_{502}, R_{300} and R_{153}. In the R_{502} and R_{300} these structures occurred in abundance, especially in the fibrotic nodules and to a minor extent in the alveoli and macrophages. No such structures could be detected in the snowit and R_0 groups. According to Jones[1] the South African gold-bearing rock contains many acicular fibres which he regarded as sericite and from which he concluded that sericite was the main cause of fibrosis. Fallon and Banting,[8] Lemon and Higgins,[10] and Cummins[7], however, found sericite to be much less fibrogenetic than quartz. The high pathogenicity of the R_{502} and R_{300} samples could therefore not only be ascribed to their possible sericite content.

King, Gilchrist and Rae[9] found that acid treatment of sericite increased its fibrogenetic properties. In spite of acid pre-treatment of Sample R_0 to remove its radioactivity, it produced less fibrosis than R_{153} over the initial 150 days. (R_0 was prepared from R_{153} by acid treatment.) The fibrogenetic activity of sample R_{153}, therefore, probably depended on its radioactivity.

It is interesting to note that there was no correlation between silica content and grade of fibrosis during the early stage of the experiment. At this stage the grade of fibrosis produced corresponded to the degree of radioactivity of the various dust samples. This appeared to be very important especially when the composition of sample R_{502} is considered. It consisted of very fine particles obtained from the original dust R_{153} (see material). This fine dust (R_{502}) contained more radioactive material and less silica and produced more fibrosis than the original. This observation might explain the extremely high pathogenicity of the Witwatersrand mine dust in the following way:

Although the natural mine dust (R_{153}) produced only slightly more fibrosis than the non-radioactive R_0 sample, it is possible that those working underground are in fact exposed to a dust with a much higher radioactivity than natural mine dust, (R_{153}). The finer the dust particle the longer it will remain airborne and the better it will be inhaled. According to Orenstein[13] we are justified in regarding with some scepticism the ability of water to remove from the air the dangerous very fine particles. Since these dust particles have the highest radioactivity the inhaled dust will be more radioactive than natural mine dust, (R_{153}).

CONCLUSION AND SUMMARY

The fibrogenetic action on the lungs of rats of different radioactive mine-dust samples was investigated. In the presence of radioactive material no correlation was found

between the silica content of the samples and the grade of fibrosis produced. During the early stages of the experiment the samples containing radioactive material caused a greater fibrosis than the non-radioactive mine dust sample. During this stage the grade of fibrosis produced corresponded to the degree of radioactivity of the samples. In the later stages no marked difference was observed. It is suggested that the presence of radioactive material in the Witwatersrand mine dust contributes to its exceptional fibrogenetic activity.

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TIBIALE VERLENGING VIR BEENVERKORTiNG *

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Beenekwalisasie is 'n onderwerp waaroor daar baie meningsverskil bestaan. Die betreklike onpopulariteit van beenverlenging dui op die mislukkings van die verlede. Verskil in beenlengtes het jare lank al 'n moeilike probleem geskep. Die verskillende faktore verantwoordelik vir beenverkorting maak 'n eenvoudige benadering tot hierdie afwyking onmoontlik. Daarby dui die groot aantal prosedures wat al aangewend is om die verkortings te oorkom daarop dat daar nie 'n eenvoudige antwoord vir die probleem is nie.

Verskil in beenlengte is betreklik algemeen. In 'n volwasse persoon is 'n verkorting van $\frac{1}{4}$ duim normaal en 'n groter verkorting word beskou as gebrekkig. 'n Verkorting van meer as 2 duim is 'n ernstige hindernis. Afgesien van die meganiese ongemak, is die sielkundige effek soms baie groot en daarby kan die individu nie op gelyke terme kompeteer op die arbeidsmark nie. Die abnormale slytasie in die normale been lei in baie gevalle tot vroeë osteo-artritiese veranderinge in die heup en ook in die rug. 'n Opgeboude skoen is ook baie duur. Beengelykmaking is 'n probleem wat dikwels voorkom en dit is van die grootste belang vir die pasiënt.

In Edinburgh, veral, is daar 'n intensiewe belangstelling in die probleem, en die tegniek wat vervolgens beskrywe word, het dáár sy ontstaan gehad. Die meeste werk i.v.m. beengelykmaking is egter in Amerika gedoen. Of gelyke beenlengtes verkry moet word deur verlenging van die korter been of verkorting van die langer been, is kontensieus.

Beginsels by die Behandeling van Beenongelykheid

Dit is moeilik om vaste reëls neer te lê waar so baie verskillende faktore in aanmerking geneem moet word, dog dit is veilig om die volgende twee reëls na te kom:

Eerstens, in 'n pasiënt met een uitgeteerde been, moet die geaffekteerde been behandel word. As daar dan onvermydelike komplikasies na die operasie mag volg, sal die pasiënt nie so baie verloor nie. Maar 'n komplikasie in die pasiënt se enigste normale been sal katastrofies wees.

Tweedens, in 'n pasiënt met, sover dit krag betref, 2 normale bene, maar een verkort, moet die langer been verkort word. In 'n lang persoon, moet verkorting oorweeg word en in 'n kort persoon verlenging.

Soos dus hiervan afgelei kan word, word tibiale verlengings meestal in ou poliogevalle gedoen. Die polio-entstof sal sal hierdie misvormende siekte hopelik iets van die verlede maak, dog die ou poliogevalle sal nog baie jare lank met ons wees.

Beskikbare Tegnieke

Die verskillende tegnieke beskikbaar vir beengelykmaking kan groepeer word onder die volgende 3 hoofde:

1. *Beenverkorting* is 'n groot operasie op 'n normale ledemaat en word gedoen vir verkorting weens 'n T.B.-heup, en vir 'n kongenitale kort been. Die been teenoor die verkorte been moet verkort word. Dit is veiliger om dit op die tibia te doen, as op die femur.

2. *Teenhouding van epifisiële groei.* Hierdie prosedure kan alleenlik in kinders met 'n groeiperiode van nog etlike jare gebruik word. Die epifisiële blokoperasie van Phemister[1] en die meer populêre gebruik van die kram (staple), [2, 3] veral in Amerika, is die 2 operasies in gebruik om epifisiële groei in die langbene te verminder. Die epifese van die onderent van die femur, aan die normale kant, en soms ook die proksimale tibiale epifese, word uitgeskakel en daardeur word die groei in die normale been verminder, terwyl die verkorte been toegelaat word om te groei, sodat die twee gelyk mag wees as volwassenheid bereik word. Dus, eerstens moet die verwagte groei van die verkorte been 'bereken' word. In polio sien ons die beeld dat hoe vroeër die ledemaat affekteer is, en hoe jonger die kind is, des te groter is die verkorting. Tweedens moet die verwagte groei van die normale been bepaal word. Soveel faktore beïnvloed beengroei dat geen presiese metode beskikbaar is om die verwagte groei van 'n been te bepaal nie. Dog tabelle is beskikbaar wat dui op die verwagte verkorting of groei, wat sal plaasvind.[4, 5] Die voordeel verbonde aan klamme is dat epifisiële groei tydelik gestaak kan word en dat verdere groei plaasvind met verwydering van die klamme. Of die groei na verwydering van die klamme normaal sal voortgaan, is te betwyfel. Alle berekenings word dan omvergewerp.

3. *Beenverlenging* kan verkry word deur stimulering van die epifisiële groei of deur meganiese distraksie. Van al die metodes om bene gelyk te maak, is groei-stimulering van die

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verkorte been teoreties die aanneemlikste, dog geen betroubare en veilige metode is egter so ver ontwikkel nie. Die soektog na só 'n metode, begin deur Von Langenbeck[6] in 1869, gaan steeds voort. Die groot getal en verskeidenheid van metodes wat al gebruik is in die verlede, wys dat die probleem nog onopgelos is. Dit is bewys dat 'n verhoogde bloedtoevoer na die lang bene van kinders, beengroei sal stimuleer. Die meerderheid van metodes poog om 'n vermeerderde bloedtoevoer na die ledemaat te kry, veral in die omstreke van die epifese. Die direkte metodes behels o.a. eenvoudige periosteale afstroping, metafisiële boorgate, en inplanting van sulke vreemde materiaal soos yster, koper, ivoor en selfs groeihormoon-poeier.[8] Die mees algemene indirekte metode in gebruik, is lumbale simpatektomie. Harris[7] beweer dat die resultate in lumbale simpatektomie teenstrydig is, en dat die effek van simpatektomie op die groei van 'n verkorte been meer van teoretiese as praktiese waarde is. Totdat 'n ideale metode van epifisiële stimulering gevind word, bly ons dus oor met die enigste praktiese alternatief—meganiese verlenging van die verkorte been.

GESKIEDKUNDIGE OORSIG VAN BEENVERLENGING

Die eerste aangetekende poging tot beenverlenging, is beskryf deur Codivilla[8] in 1905. Putti,[9] in 1921, was die eerste persoon wat die nou aanvaarde beginsel van traksie en teentraksie direk aan die verkorte been gebruik het.

In 1927 het Abbott[10] 'n metode van tibiale verlenging beskryf, wat met modifikasies 'n standaardoperasie in Amerika geword het. Abbott se operasie vorm die basis van die operasie wat ons nou nog doen. Abbott, na hy die tibia ingesny het, het traksie en teentraksie aangewend deur middel van Steinmann-penne proksimaal en distaal van die osteotomie. Die stappe van sy operasie was: (1) Verlenging van die tendo achilles, (2) osteotomie van die fibula, (3) die Steinmann-penne is aangebring, (4) osteotomie van die tibia en insnyding van die periosteum van die tibia reg rondom die been, asook van die diep fascia op die antero-laterale aspek van die been en (5) die distraksie apparaat is daarna aangebring. Die beenente is dan geleidelik van mekaar getrek.

Abbott het na-operatiewe moeilikheid gehad om voorwaartse angulasie van die fragmente te voorkom weens die boogsnaaraksie van die gespanne spier. Baie veranderinge is al aan die apparaat aangebring o.a. ook om hierdie moeilikheid te oorkom, want dit is hoofsaaklik hiér waar hy gefaal het.

Kontraindikasies vir Beenverlenging

1. Die ouderdom van die pasiënt. Volgens Campbell[5] moet slegs jong volwasse persone met 'n korting van 2 duim of meer, as geskikte persone beskou word. Dit is geen wonder dat hulle dan besluit dat ‚in ons ondervinding, is beenverlenging 'n uiters gedugte onderneming wat 'n lang periode van ongeskiktheid insluit met baie lyding, en dit is selde geregverdig'. *Dit* skyn die algemene opvatting by die meeste Amerikaanse chirurge te wees wat die Princess Margaret Rose-Hospitaal in Edinburgh besoek, waar al meer as 150 verlengings gedoen is. Ons volg die tegniek van tibiale verlenging soos ontwikkel deur drs. Anderson en Stirling in Edinburgh. As die verkorting van so 'n aard is dat meer as 2 duim van verlenging nodig is, mag die operasie nog vroeër gedoen word, sodat in uitsonderlike gevalle 'n tweede operasie 'n paar jaar later gedoen kan word. Beenverlenging mag tot die ouderdom van 14 jaar gedoen word met omtrent geen pyn nie. Tussen 14 en 16 jaar moet die operasie selde gedoen word en word gewoonlik vergesel van 'n dowwe pyn, wat selde pynstillers benodig. In 'n volwasse persoon is die prosedure moeiliker, meer geneigd tot senu- en vaskulêre komplikasies en baie pynlik.

2. Onlangse of aktiewe been- of gewrigsontsteking.

3. Korting wat so groot is dat die aanbring van 'n kunsbeen beter sou wees.

4. 'n Lang persoon in wie verkorting van die ander been oorweeg moet word.

Femorale Verlenging

Hierdie prosedure is geensins populêr nie. Meganiese verlenging van die femur is baie moeiliker en permanente of tydelike beperking van kniebewegings word in alle geval gesien. Die styfheid word verklaar deur die pen wat deur die onderste derde van die femur geplaas word, asook weens die geweldige druk wat op die kondiles uitgeoefen word.

Femorale verlenging met 'n beenskyf wat tussen die beenente geplaas word, na verdeling van die femorale skag, is ook nie in algemene gebruik nie. Dié beenoorplanting neem ongeveer 'n jaar om vervang te word.

Tibiale Verlenging

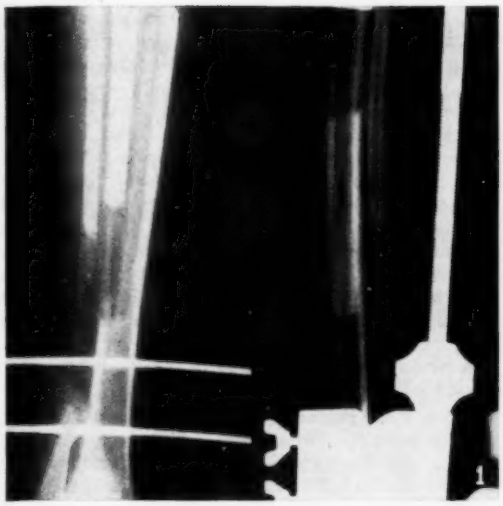
Hierdie prosedure word meer geredelik aanvaar. Daar is goeie redes waarom dit 'n beter prosedure is. In polio vind meeste van die beenverkorting in die tibia plaas en tibiale torsie kan gedurende die verlenging gekorriseer word. Afgesien daarvan dat tibiale verlenging 'n makliker operasie is, kan kniebewegings gedurende die hele verlengingsperiode gedoen word.

Die hoofindikasie vir beenverlenging is beenverkorting van meer as 1½ duim. Vir minder verkorting, buiten as die heup of rug styf is, word gekompenseer deur bekkenoorhelling en/of skoenopbouing. Volgens Ingram,[5] is absolute gelykmaking nie altyd wenslik nie. 'n Pasiënt wat 'n lang kaliper moet dra is gewoonlik beter daaraan toe met 1 of 2 cm. verkorting aan die geaffekteerde kant. As 'n lang kaliper nie benodig word nie, moet die verlenging absolute gelykmaking verkry. As voetstabilisasie later beplan word, moet 1 cm. by die verlenging gevoeg word, om te kompenseer vir die vermindering in lengte wat die voetoperasie teweegbring.

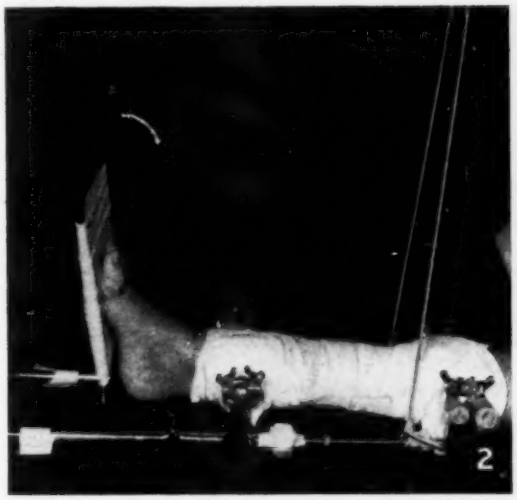
Tegniek van Tibiale Verlenging

Die Eerste Stadium van beenverlenging behels 'n osteotomie van die fibula ongeveer 1½ duim bokant die laterale malleolus en oorplanting van die distale fragment in die tibia (Afb. 1). Die proksimale fragment met periosteum word verwyder (ten minste ½ duim) om te verhoed dat die fibula weer aangroei, wat dit andersins geredelik doen. Hierdie verandering in die Abbott-tegniek is ontwikkel deur Anderson[11] en verhoed die valgusmisvorming wat so algemeen na die oorspronklike Abbott-operasie was. As die laterale malleolus nie goed vasgehou word nie, trek dit gedurende die verlenging na bo en kom te lê teenoor die distale tibiale epifese. Die prikkeling daardeur veroorsaak, verminder die groei aan die laterale kant van die distale tibiale epifese, en dit lei dan tot 'n valgusmisvorming van die voet.

Allan[12] het opgemerk dat subluksasie van die fibulêre kop in heelparty van sy gevalle voorgekom het. Hy meld ongelukkig nie hoe hy die fibula afgesny het nie, dog dit skyn eenvoudig osteotomies te gewees het, sonder oorplanting na die tibia. In meer as 150 gevalle in Edinburgh, is subluksasie van die fibulêre kop slegs in een geval gesien—'n



Afb. 1. A.P. en laterale opname met masjien in posisie. Die A.P.-plaat toon die osteotomie van die fibula. Ou poliogeval, 9 jaar oud, met 2½ duim verkorting; 2½ duim verlenging is gedoen, asook 40° derotasie vir eksterne rotasie van tibia.



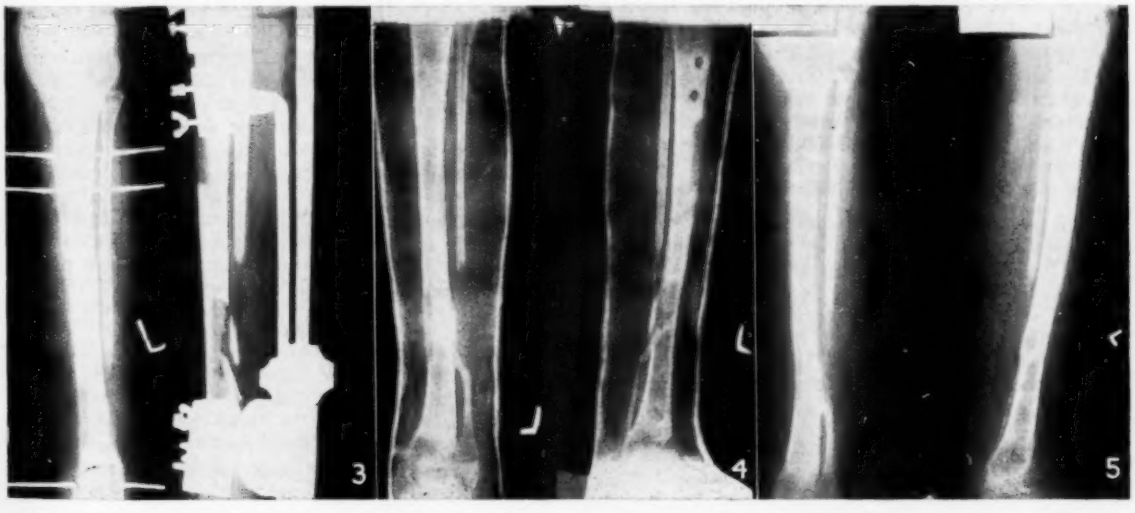
Afb. 2. Foto toon die verlengingsapparaat in posisie ongeveer 2 duim bokant die komberse.

geval waar die fibulêre osteotomie onvolledig was en die daaropvolgende beenhegting nie opgemerk is nie.

Allan het 'n hele paar gevalle gehad met tydelike peroneale senuverlamming, en ek is geneig om te dink dat hierdie komplikasie by gevalle met subluksasie voorgekom het, of andersins was hulle te wyte aan die gips-silinders wat hy gebruik aangesien hierdie komplikasie seldsaam is.

Die Tweede Stadium volg 6 weke later. Die beenverlengingsapparaat of ‚masjien' wat gebruik word is basies 'n Abbott-masjien, met Young se modifikasies. Die masjien word slegs aan die tibia gefikseer en word dan 2 duim bokant die komberse gehang, vanaf 'n Balkan-raam (Afb. 2). Die knie- en enkelgewrigte is los om te kan beweeg. Kortliks word die operasie as volg gedoen:

Die penne word aangebring met 'n elektriese automatiese boor, met al 4 penne in dieselfde vlak. 'n Spesiale voerder is beskikbaar. As die boonste pen, die eerste een, aangebring word, word die vel distaalwaarts getrek. Die pen word vanaf die laterale kant ingesit, en die pen, nadat dit deur die vel gesteek is, gaan eers onderhuids, om te verhoed dat die laterale popliteale senu beskadig word. Dan word die spiere versigtig teruggedruk met die pen, sodat dit die tibia teen 'n reghoek binnegaan ongeveer 1½ duim onderkant die proksimale tibiale epifese in die koronêre vlak.



Afb. 3. Ou poliogeval, 16 jaar oud, met 2½ duim verkorting van linkerbeen; 2 duim verlenging gedoen.
Afb. 4. Plate van dieselfde pasiënt as Afb. 2. Die been is in gips en goeie belyning is teenwoordig.
Afb. 5. Dieselfde pasiënt as Afbs. 2 en 3. Die been is uit gips uit 4 maande na die verlengingsperiode. Goeie kallus is teenwoordig.

Die distale pen word net bokant die distale tibiale epifese geplaas, nadat die vel eers opgetrek is. Die ander 2 penne word dan soortgelyk ingesit en die masjien aangebring en vasgedraai.

'n Reguit lengtesnit word dan gemaak oor die voorste aspek van die tibia, die periosteum ingesny en losgewerk. Die periosteum word dan sirkulêr ingesny om pyn gedurende verlenging te voorkom. Die been word dan òf met 'n Z- òf skuinsosteotomie deurgesny. Eersgenoemde verdeling is meer populêr as laasgenoemde. Die osteotomie-eindes moet meer as twee keer so lank as die beoogde verlengingsafstand wees. Indien eksterne rotasie teenwoordig is, kan dit op hierdie stadium gekorriseer word deur die masjien te stel. Die periosteum en vel word geheg en droë verbande aangebring oor die wond en rondom die pengaatjies.

Die na-operatiewe behandeling is waarskynlik die belangrikste aspek van tibiale verlenging. Verlenging begin na 48 uur, en geskied teen 1/16 duim per dag, totdat die beoogde lengte bereik is. As die pasiënt pyn ondervind word die spanning met 1/16 duim verslap en die apparaat word 3 tot 4 dae lank nie gedraai nie. Fisioterapie vir die knie en voet begin saam met die verlenging. Die verbande word 2 weke lank ongehinderd gelaat, behalwe as infeksie byvoorbeeld vermoed word. Die steke word dan verwyder, en die pengaatjies weekliks skoongemaak. As die verlangde afstand bereik is, word die masjien so gelaat tot genoeg kallus gevorm het. Dit mag 3 tot 6 weke duur. Dan word die masjien verwyder en plankies oor die penne aangebring, mediaal en lateraal, om hulle in posisie te hou. Dan word 'n vollengte-gips aangebring, wat al die penne insluit. Die voet word in maksimale dorsifleksie geplaas. Die pasiënt word dan ontslaan met krukke. As beenhegting sterk genoeg is, word die gips en penne verwyder. Nog 'n vollengte-gips word aangesit totdat beenhegting solied is. As 'n equinusmisvorming as gevolg van die verlenging ontstaan, kan dit grootliks oorkom word, deur die gips oor die dorsum van die voet te verwyder, en die voet op te wig deur stukke velt onder die voorvoet in te druk. As die equinusmisvorming te groot is, moet 'n tendo achillesverlenging gedoen word. Die neiging is om nie 'n primêre tendo achillesverlenging te doen nie, dog om te wag tot na die verlenging om te sien hoe groot die misvorming dan is. Die presiese tendo achillesverlenging kan dan beter bepaal word.

KOMPLIKASIES VAN BEENVERLENGING

Komplikasies was die hoofhindernis in die verlede. In 1936 het Compere[13] die saak vir en teen beenverlenging gestel. Sy kritiek oor hierdie operasie is oor en oor gebruik deur verskillende skrywers om 'n operasie wat sedertdien betreklik veilig geword het, te verdoem. Ek wil die punte deur hom genoem herhaal: (1) Komplikasies as gevolg van oorrekking, (2) komplikasies as gevolg van steuring van die bloedtoevoer na die fragmente en (3) komplikasies weens onvoldoende fiksering van die fragmente.

Die laaste 2 komplikasies kan oorkom of verhoed word deur veranderinge in die operasietegniek en die verlengingsapparaat. Tot 1936 is beenverlenging gedoen, volgens Abbott se beskrywing daarvan. Die metode het insnyding van die diep fascia van die kuitarea behels met wye blootlegging van die weefsels. Dit het ongetwyfeld die bloedtoevoer na die fragmente verminder.

Ek wil komplikasies as gevolg van oorrekking meer breedvoerig bespreek. Teen verlenging van 1/16 duim per dag word geen beenkomplikasies gesien nie. Niehegting word nooit gesien nie, alhoewel vertraagde hegting af en toe gesien word. Dit is egter van korte duur. Ferme beenhegting is waarneembaar ongeveer 3 tot 4 maande na die verlengingstydperk. Infeksie langs die pen kom selde voor en is gewoonlik lig en klaar vanself op. Selde is daar 'n geval wat skraping van die sinus benodig om die infeksie op te klaar. Daar is relatief gesproke geen spierkomplikasies nie, alhoewel 'n ligte mate van spierverswakking verwag kan word. In baie gevalle is daar voor die operasie in elk geval minimale spierkrag teenwoordig. Allen[12] het 'n groot mate van spieratrofie in sy gevalle gesien—dit was ongetwyfeld te wyte aan sy distraksieapparaat wat die been in gips gehou het vanaf die tone tot by die heup. Dit het geen knie- of enkelbewegings toegelaat nie.

Die bloedvate rek verbasend maklik en wys nooit enige tekens van vaskulêre ooreising nie.[12] Die senus is die enigste belangrike strukture wat somtyds moeilikheid gee. Die pasiënt merk af en toe 'n ligte mate van hipoëstesie oor die dorsum van die voet. Dit is egter tydelik van aard.[12]

Die enigste komplikasie wat in aanmerking geneem moet word, is voet- en enkelmisvormings. Die voet neig om in equinus te gaan weens die spanning in die tendo achilles. Dit kan gekorriseer word deur wigting of verlenging. Die tone neig in fleksie waar die dorsifleksors verlam is, en die ekstensors in effe dorsifleksie waar die toonfleksors verlam is. Dit is egter tydelik van aard en klaar geheel en al op met fisioterapie. Die enkelgewrig neig om 'n klein mate van beweeglikheid te verloor. Hierdie is die enigste komplikasie wat van belang is, en genoem behoort te word. Die beperking van beweging is van so 'n geringe aard dat dit nie oorbeklemtoon moet word nie. Die velwonde gee nooit moeilikheid nie, weens die feit dat 'n reguit vertikale snit gebruik word in teenstelling met Abbott se krom velsnit. Die sirkulasie van die voet bly goed gedurende verlenging en trombose is ongekend. Laat komplikasies word nie gesien nie.[14]

Komplikasies soos pyn, vaskulêre en senuoorstremming behoort nie gesien te word nie, as die regte tegniek gebruik word en as 'n pasiënt in die regte ouderdomsgroep gekies word. Die prosedure van tibiale verlenging behoort meer dikwels gedoen te word, dog verkieslik in 'n groot hospitaal waar spesiale personeel beskikbaar is vir ortopediese verpleging. Ons het nou al 5 gevalle sonder enige komplikasies in die Lady Michaelis-Ortopediese Tehuis gedoen. Die verlengings het gewissel van $1\frac{1}{2}$ tot $2\frac{1}{2}$ duim. Ons is baie bemoedig deur die korttermynresultate en is van plan om die prosedure meer dikwels uit te voer.

OPSOMMING

1. 'n Kort oorsig van metodes van beenverlenging word beskryf.

2. Spesiale aandag word gegee aan tibiale verlenging volgens 'n Abbott-tipe operasie en verlenging.

3. Komplikasies wat vroeër algemeen was na tibiale verlenging, is nou uitgeskakel met 'n veranderde tegniek.

4. Die vooroordeel teen die prosedure behoort nou iets van die verlede te wees en die operasie behoort meer dikwels gedoen te word.

SUMMARY

1. A short review of the methods used in lengthening of limbs is given.

2. Special attention is given to tibial lengthening according to an Abbott type of operation and lengthening.

3. Complications which commonly occurred previously after tibial lengthening are now eliminated as the result of a new technique.

4. The prejudice against the procedure should now disappear and the operation should be performed more often.

Ek wil die Mediese Superintendente van die Karl Bremer-Hospitaal, Bellville en die Lady Michaelis-Ortopediese Tehuis, Plumstead, en dr. Hamilton Bell, hoof van die Afdeling Ortopedie bedank vir hul toestemming tot publikasie van hierdie artikel. Ook my hartlike dank aan dr. J. C. S. Steytler vir die deurlees en advies oor die artikel. Ander persone wat ek spesiaal wil bedank, is drs. Robert Stirling en W. V. Anderson, albei van Edinburgh, wat my die tegniek van tibiale verlenging, soos deur hulle ontwikkel, geleer het.

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DIE ONTWIKKELING VAN DIE REGOPSTAND VAN DIE MENS*

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Presies 100 jaar gelede (1850) het Charles Darwin die evolusiebegrip die eerste keer op 'n wetenskaplike basis geplaas. Sy idees omtrent die evolusie van die mens maak hy eers in 1871 bekend. Een van die aspekte waaraan hy sy aandag wy is die ontwikkeling van die regopstand by die mens en die groot moontlikhede wat dit inhou vir die vrygeworde hande. In sy boek *The Descent of Man* skryf hy:[5] ‚I can see no reason why it should not have been advantageous to the progenitors of man to have become more and more erect or bipedal' (p. 78), en 'n entjie verder: ‚As the progenitors of man became more and more erect . . .'

Myns insiens het Darwin hom nie 'n halfregop tussenstadium in die menslike ontwikkeling voorgestel nie, want hy skryf: ‚It might have been argued with great force and apparent truth, that an animal could not have been gradually converted from a quadruped into a biped, as all the individuals in an intermediate condition would have been miserably ill-fitted for progression.' Hy het meer 'n stadium in gedagte soos verteenwoordig deur die mensape wat normaalweg vierbenig voortbeweeg met die romp taamlik regop en die kneukels op die grond, òf heeltemal regop soos *Hylobates* (die Gibbon) wat regop kan loop sonder om dit te leer, soos Darwin dit stel.

DIE LA CHAPELLE-GERAAMTE

Vyftig jaar gelede (1908) is die eerste taamlik volledige menslike fossielreste by la Chapelle aux Saints gevind. In 1911-13 beskryf Boule[2] hierdie skelette en tot vandag geld die bene van die sogenaamde ‚ou man' as die tipe van *Homo neanderthalensis* wat die rekonstruksie van die geraamte betref. Boule het na 'n baie volledige ondersoek van die la Chapelle-eksemplaar gevind dat daarin 'n aantal aapagtige kenmerke voorkom. Wat die stand van die individu betref het hy veral klem gelê op die volgende:

1. Die planum nuchae is meer skuins en die condylus occipitalis en foramen magnum is meer dorsaal op die skedelbasis geleë as by die moderne mens. Dit sou beteken dat die kop na vore geknik was.

2. Die (onderste) nekwerwels verskil baie van dié van 'n hedendaagse mens. Die processus spinialis is lank, horisontaal en ongevurk soos dié van 'n sjimpansee en die zygopophyse is minder skuins. Die werwelkolom is dus gerekonstrueer met slegs die primêre kurwe soos by die sjimpansee.

3. Die femur is sterk gekrom en die kop van die tibia wys 'n aansienlike mate van retroversie. Dit sou daarop dui dat die Neanderthal-mens met gebuigde knieë gestap het.

Hierdie rekonstruksie van Boule van die la Chapelle-geraamte in die ‚bekruipende' houding is tot met die nuutste uitgawe van sy boek deur Vallois[3] (Engelse uitgawe 1957) herhaal en is in talle werke oorgedruk.

KRITIESE BENADERING

Die feit dat hierdie voorstelling van Boule sonder meer deur die meeste antropoloë aanvaar is, moet in 'n sekere mate gewyt word aan die geestelike klimaat van daardie tyd. Die idee was natuurlik om die Neanderthal-mens so aapagtig as moontlik voor te stel in hulle soektog na 'n vermiste skakel. Darwin se waarskuwing dat 'n halfregop wese 'n geheel onmoontlike houding het, is eenvoudig in die wind geslaan. Ook is geglo dat met die uitsondering van die Gibbon, die mensape ook nie volkome regop is wanneer hulle tweebenig voortbeweeg nie. So skryf Slijper,[8] wat so 'n groot bydrae tot die algemene bewegingsleer gemaak het, in 1947 nog: ‚De anthropomorphe apen . . . zein echter in staat op de achterbeenen alleen te lopen; de romp helt daarbei egter sterk naar voren.'

Die mens is nie die enigste dier wat tot 'n tweebenige manier van voortbeweging oorgegaan het nie. By die tweebenige reptiele word die romp natuurlik nie volkome regop gehou nie, maar die houding is nie onmoontlik nie omdat die lang en soms baie swaar stert dien om die liggaam te balanseer. By die voëls wat met skuins rompe loop (ganse, swane, ens.), is die nek gewoonlik lank en word vér na agter gehou om die swaartepunt na agter te skuif. By voëls met 'n betreklike regopstand (bv. roofvoëls) is die nek kort. Nêrens in die diereryk word 'n dier aangetref waarvan die swaartepunt voor die steunpunt lê wanneer hy normaalweg voortbeweeg nie.

Coon[4] (1954) wys ook daarop dat van alle mensfossiele wat tot dusver gevind is, slegs die Neanderthal-mens nie regop geloop het nie. Die sleutel tot die probleem het dus

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daarin gelê om te ondersoek of die klassieke rekonstruksie van die Neanderthal-mens geregverdig was of nie.

Geheel onafhanklik het 'n aantal ondersoekers dié aspekte van die skelet van die Neanderthal-mens ondersoek wat betrekking het op sy stand. So kom Arambourg[1] (1955), Patte[6] (1955), Straus en Cave[9] (1957) en ekself[10] (1957) tot die gevolgtrekking dat wanneer die foute wat gemaak is as gevolg van die patologiese toestand van die oorblyfsels (Straus en Cave) en die foutiewe rekonstruksie van die planum nuchae buite rekening gelaat word al die kenmerke van die Neanderthal-mens binne die variasiewydte van die moderne mens val. Selfs die nekwerwels waaraan so baie waarde geheg is, regverdig nie 'n stand anders as dié van die hedendaagse mens nie.

GEVOLGTREKKING

Die Neanderthal-mens het dus regop geloop soos al die mense van wie fossielreste tot dusver ontdek is, insluitende die Australopithecinae.

Die feit dat die Neanderthal-mens regop moes geloop het, is reeds in 1955 deur Schultz,[7] die bekendste lewende primatoloog beklemtoon. Hy skryf: ‚I could never understand how any creature can be *nearly upright* in the manner in which Neanderthal man has sometimes been reconstructed with his trunk leaning forward and taking a long step in order to prevent his falling.' (Schultz verwys hier klaarblyklik na Weinart (1947) se rekonstruksie van die la Chapelle-mens.) Hy gaan ook verder en gee 'n aanduiding van die ontwikkeling van die regopstand. Die halfregop houding waar die swaartepunt voor die steunpunt lê, word nooit ingeneem deur 'n kind wat leer loop of 'n aap wat op sy agterpote staan nie. Selfs in die beginstadia van die regopstand moes die romp ten volle regop gehou word om gebalanseer te wees, en hierdie stand verander dan geleidelik van 'n sporadiese poging tot 'n permanente gewoonte.

CONCLUSION

Neanderthal man walked erect like all the human beings of whom fossil rests have thus far been discovered including the Australopithecinae.

The fact that Neanderthal man must have walked erect has already been pointed out by Schultz, the most well-known primatologist, who, in 1935, wrote as follows: 'I could never understand how any creature can be *nearly upright* in the manner in which Neanderthal man has sometimes been reconstructed with his trunk leaning forward and taking a long step in order to prevent his falling.' (Schultz is here obviously referring to Weinart's (1947) reconstruction of the la Chapelle man.) He goes further and indicates the development of the erect posture. The semi-erect posture, where the centre of gravity lies in front of the fulcrum is never adopted by a child who is learning to walk or an ape standing on its hind legs. Even in the early stages of the erect posture, the trunk had to be held completely erect in order to maintain its balance and this posture gradually changed from a sporadic attempt into a permanent habit.

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A PAGE OF DURBANVILLE HISTORY *

G. M. MALAN, M.B., CH.B., *Durbanville, President, Cape Western Branch, Medical Association of South Africa*, 1958

Durbanville was one of the first outer areas to be settled outside the Peninsula proper and so we have farms in the Tygerberg called van Riebeeck's Hof, said to have been used by Johan van Riebeeck as a cattle station; Maastricht, a farm owned by Governor Jansens and called after his home town in Holland; and Vissershok, a farm along the Diep River on the Malmesbury Road, which has the date 1768 on its gable. Vissershok was the first farm in South Africa where wheat was sown. It is the place where fishing boats used to anchor a safe distance from the mouth of the river at Milnerton. This Diep River has its source at Durbanville and joins the Malmesbury river near Philadelphia to empty into the sea at Milnerton. The hill above Durbanville is called Kanonkop and there one of the cannon is still to be seen which was used to signal to farmers in the area that ships had arrived in the harbour and that they could bring their produce. This cannon is now the corner post for the fences of 4 farms which converge there. A most marvellous view is to be seen from this hill on a spring day. Durbanville, lying at its foot, was first called Pampoenkraal and then D'Urban after Sir Benjamin D'Urban, but this name was later changed to Durbanville to prevent confusion with the city that was growing in Natal.

Dr. Malan

In his *History of Medicine in South Africa* (p. 129) Dr. E. H. Burrows gives a photograph of Dr. F. L. C. Biccard (1809-1884), a son of Dr. L. G. Biccard (1775-1822), the first doctor officially licensed to practise in South Africa (p. 73). Dr. F. L. C. Biccard was the first man to use chloroform at the Cape for anaesthesia; this was in 1850 (p. 129). In 1866 Juta published his *Volksgeneeskunde voor Zuid-Afrika*, one of the first medical books to be published in South Africa; he practised at Durbanville, Cape Town and Malmesbury and later was in charge of the Infirmary at Robben Island. In the smallpox epidemic of 1840 he conducted a lazaretto at Tygerberg. Today there still lives at Durbanville, now in his 88th year, Dr. L. F. B. Biccard—a retired practitioner and grandson of Dr. F. L. C. Biccard.

The district has an interesting link with Napoleon in that one of his aides, Count de las Cases was kept under custody at the farm Tygerberg. This farm is now called Altydgedacht, and has been owned for the last 106 years by the Parker family. On this farm I discovered *The Memoirs of the Life, Exile and Conversations of the Emperor Napoleon* by the Count de las Cases, in 4 volumes. Published in New York in 1855, these memoirs tell how the Count (vol. iv., p. 227) 'was separated from the person of General Bonaparte on St. Helena because he corrupted the fidelity of an inhabitant of the Island, so far as to render him, in a criminal and underhand way, the bearer of secret and clandestine letters for Europe', and in conformity with the directions of the British Government 'will be transported to the Cape'.

He left the Island on 31 December 1816 and arrived at the Cape on 17 January 1817, and the ship put in to the harbour at Cape Town and not at Simon's Bay, as had been planned, because of

* Valedictory Presidential Address, Cape Town, 30 January 1959.

the violent south-easter which was blowing. The Count describes the clouds coming over the mountain 'vulgarly called spreading the table cloth'. He speaks of the beautiful harbour. After 2 days he was lodged in the Castle and Dr. Barry came to attend him because he was said to be very ill. The Count was continually writing letters to the Governor, Lord Charles Somerset, and seems to have been a very difficult man to placate. He complains of the dungeon in which he is lodged, and that officers came to sit at his table, forcing him to take his meals in bed. However, on 29 January Lord Charles Somerset took a trip into the interior and the Count was allowed to take up residence at Newlands in the Governor's country house. This place, where he stayed until 8 April, he describes as a paradise after prison. Neighbours called and officials took notice of him. He sent to Emperor Napoleon a few articles of which he stood in need, including 'Constantia wines'. Then the Governor returned and he was sent to another place '8 or 10 leagues from the Cape' called Tygerberg, owned by Mr. Baker, a native of Coblentz.

'... We looked on him (Mr. Baker) as one of our own countrymen from his origin, his opinions, and the sympathy he showed us. The family altogether formed the most agreeable society imaginable. It would have been impossible for us to have been treated with greater respect and attention. We now commenced the third period of our captivity at the Cape. The first was our insupportable imprisonment in the Castle, which fortunately lasted only 10 days; the second was our abode in the charming retreat of Newlands; the third was our residence at the desert of Tygerberg, which was to continue for the space of 4 months. At Tygerberg we found ourselves situated on the confines of the wandering hordes. The country here and there presented detached habitations at considerable distances from each other. These were occupied by cultivators of various nations who were clearing the new grounds in order to turn them to profitable account. Though now removed to the very extremity of the civilized world, we found ourselves treated with even more than kindness. The people were neither ignorant nor indifferent with respect to the events of Europe. The majority of the population was Dutch and was therefore connected with our national system. Thus to my surprise I found the name of Napoleon familiar in this desert. The most victorious game cock in the neighbourhood was called Napoleon! The swiftest race horse was Napoleon! The most invincible bull in the country was Napoleon! I could not refrain from laughing at this. But everyone has his own way of rendering honour and respect; and Napoleon was the noblest of all names in the estimation of the inhabitants of Tygerberg'.

Page 281: 'During the increasing indisposition under which I was labouring at Tygerberg, I wrote to the Governor to permit me to return to the town for the sake of obtaining medical aid. But this request was in vain; Lord Charles Somerset now turned a deaf ear to my representations. In the impatience and irritation excited by the prolongation of my captivity I several times during my residence at Tygerberg resumed, and perhaps in energetic terms, my appeals to the Governor requesting him that he would permit me to return to Europe.'

Page 283: 'I addressed a letter to him describing the state of my health and urging the indispensable necessity of my removal to town. But to this he coolly replied that though he could not alter his arrangement he had given orders for my obtaining medical attendance. But I was situated at a distance of 10 leagues from Cape Town; the doctor could only visit me once a week; and as he prescribed remedies which could only be procured in the town their application was impracticable. I lost all patience on reading this answer, which seemed to be an act of cruel irony rather than a measure of relief; and I indignantly addressed a letter to the Colonial Secretary. As it was by his discretion that I had been removed to Mr. Baker's, I took the liberty of informing him that, as it was absolutely necessary that I should be near my medical attendant, I presumed he would have no objection to my removal to the town to the house of Dr. Liesching, Mr. Baker's father-in-law. He replied that he had consulted with the Governor and that His Excellency had declared that his instructions did not admit of my coming back to Cape Town. But I was determined not to be satisfied with this and I again wrote to the Colonial Secretary, "That in spite of his letter nothing but absolute force should prevent me from quitting Tygerberg". Fortunately the permission for my departure at length arrived from England, just at the very moment when I was about to execute my determination. The Governor communicated this welcome intelligence to me accompanied by the offer of a lodging prepared for me in the town. But I declined accepting it and proceeded as I had originally intended to the house of Dr. Liesching, where I experienced all the attention and hospitality of Tygerberg in a truly patriarchial family whose society produced a beneficial effect on my health and spirits'.

Page 284: 'Now commenced a new series of vexations. The Governor mentioned two opportunities presenting themselves for my departure. I replied that the speediest would be the most reliable. I confidently expected the receipt of my passport. I was confined to bed. Two days elapsed and one of the ships sailed. A large transport was lying in the harbour, which was destined to convey a regiment of artillery to England. I begged the Governor to allow me to take my passage in this vessel as there were medical officers on board. I was informed that the ship was to touch at St. Helena and that this precluded my sailing in her. I had to travel by the only choice left me, an extremely small brig. The Captain informed me that he had had instructions from the Governor to prohibit me from having any communication on shore, if he should be obliged to touch at any place in the course of the voyage, and on reaching England he was not to suffer me to land until he should receive orders from the Government. I wished to obtain confirmation of this, but as it may be supposed, I received my passports and nothing more.'

Page 285: 'Towards evening on 20 August we proceeded to the beach accompanied by our two excellent hosts of Tygerberg and the Cape. The weather was calm but as we entered the boat a favourable breeze sprang up as by enchantment. We all exclaimed that this was an auspicious omen, but it was far from proving so. Our passage turned out to be one of the longest and towards the end most frightful and terrible. We got on board, the anchor was weighed and we at last set sail for that Europe we had so long wished for.'

These quotations give us some idea of the unhurried times when one could write such lengthy letters and descriptions, and tell somewhat of conditions in this district which then appeared to the European Count so much of a desert although he was treated so hospitably by the inhabitants of Tygerberg. The indisposition complained of so bitterly seems to have been psychological. He tells of profound melancholy, violent pains in the stomach, restlessness at night and pain in the head. The medical faculty could not help. After his return to France he experienced a considerable improvement in health.

Thus the story ends happily for him but he did not cease to write letter after letter to all crowned heads and heads of Governments telling of the suffering of 'one of the Lord's annointed on St. Helena'.

Madeleine Masson in her *Birds of Passage*, devotes a chapter to this same Count de las Cases, and quotes almost word for word from this Memoir. She says that nobody has ever understood why this champion of the Emperor chose to leave St. Helena rather than be isolated from his hero Napoleon on the Island. Perhaps he thought that he could be of more use to him as a free man. He had been given the option by the Governor of St. Helena.

PARLIAMENTARY NEWS : PARLEMENTÊRE NUUS

BY OUR PARLIAMENTARY CORRESPONDENT

Artificial insemination, mental illness, medical aid schemes, the cost of drugs and the care of the aged were some of the more important aspects of medicine recently discussed during a debate in the House of Assembly.

The debate was initiated by Dr. O. L. Shearer, M.P. for Pietermaritzburg City, who moved a private member's motion calling for a review of the Union's health policy with special reference to the encouragement of organizations to provide curative services; promotive health services—particularly food, housing, recreation and the care of the aged; better mental health facilities; and the better coordination of research.

Both Dr. Shearer and his seconder, Dr. Z. J. de Beer, M.P. for

Maitland, stressed that they were not pleading for socialized medicine which, they said, was repugnant to the medical profession because it restricted the medical practitioner's freedom of action and because it undermined the traditional doctor-patient relationship.

Dr. de Beer, however, made the reservation that for practical reasons socialization was unavoidable in the treatment of very poor people who could not contribute towards the cost of their medical treatment. This did not mean that they should receive inferior treatment. On the contrary it should be a matter of pride to see that these people were provided with treatment of the highest standard.

Dr. Carel de Wet, M.P. for Vanderbijlpark, was especially concerned about the alleged existence of a monopoly over the provision of ethical products and medicines. 'In all fairness I must say that to date I have been unable to determine whether the trade or the public have been detrimentally affected, but it is quite certain that this position is undesirable and fraught with very great danger', said Dr. de Wet. He urged that the Government should consider the desirability of investigating the position under the Monopolies Act.

Dr. E. L. Fisher, M.P. for Rosettenville, thought it was time for the Government to introduce legislation to control artificial insemination.

All the speakers asked for a commission to investigate health matters. Dr. Shearer wanted the commission to have very wide terms of reference to investigate the high cost of curative services, maternity services, infant welfare, health education, nutrition, housing, with special reference to environment, the potential influence of fall-out through nuclear weapons and the disposal of the by-products of nuclear science, mental health, industrial and occupational health, alcoholism, and the care of the aged.

Dr. de Wet wanted the terms of reference to 'provide for the investigation of all the various steps which will have to be taken to ensure full medical care and security for the patient'.

Dr. Fisher wanted the commission to inquire into medical aid schemes, the control of artificial insemination, the cost of drugs and the breaking down of monopolies, ways of increasing the number of doctors qualifying each year, and the breaking down of the 'division between those patients treated in mental hospitals and those treated outside these hospitals'.

Replying to the debate the Minister of Health, Dr. Albert Hertzog, said the Government realized the serious consequences artificial insemination might have—'consequences in regard to the legitimacy of a child and also repercussions in regard, for example, to incest, and that it is not impossible that within the near future we might find that a brother might marry his sister'. He assured the members of the Assembly that 'the first steps have already been taken to deal with the problem'.

Dr. Hertzog said that the terms of reference for a commission of inquiry suggested by Dr. Shearer were too wide and would lead to transgressions into the spheres of other departments over which he had no say. 'Instead of having such a comprehensive inquiry we should rather confine ourselves to those problems mentioned by the honourable member for Rosettenville (Dr. Fisher),' he said. 'Let us rather devote attention to specific problems and, where necessary, investigate them.'

Recent developments in the treatment of mental illness have made it possible to cure many patients formerly thought to be incurable. Of the 28,287 people admitted to mental institutions between 1951 and 1957, 25,613 were discharged and it is still hoped that some of the remainder will be discharged later. Manic-depressive psychosis, involution melancholia, and schizophrenia were some examples of mental diseases formerly thought incurable but which had yielded to some degree to modern treatment.

The availability of curative treatment necessitated a new approach to the problem of mental illness. Patients previously regarded as so dangerous that they were confined in straight-jackets were today allowed freedom of movement. The vital factor, however, is timely treatment. The longer the delay the more difficult the problem is. It is no use waiting until the patient becomes so ill that he has to be certified. The problem is to make drugs and treatment available to the mental patient in his immediate vicinity.

'The solution to this problem can only be found if the institutions where a man can receive treatment are near to him and today our hospitals are such institutions,' said Dr. Hertzog. 'We should try to work fast in the direction of giving the hospitals themselves the opportunity to treat people who start showing symptoms of mental disease, either through having a separate section in that hospital or through giving them separate medical treatment. These facilities need not be limited to the ordinary hospitals but these hospitals are the ideal institutions.

'It is interesting to note that a little while ago the British Commission of Inquiry, which investigated these same problems, issued their report and that the conclusions they arrived at were precisely the same as those I have been saying here. If there are large numbers of mental cases we shall have to consider establishing small hospitals solely intended for their treatment but we can consider that at a later stage.

'I therefore repeat that as a result of the new developments in our own country in recent years I agree with the honourable member (Dr. Fisher, who pleaded for the treatment of suitable mental cases in ordinary hospitals) to a large extent and I want to assure him that this problem will always have my thorough attention.'

Dr. Hertzog attributed the high cost of medical services to three main causes. The first was the monopolistic tendency. This aspect would receive serious attention. The second was the growing tendency to consult specialists. The third factor was the vast amount of research required to produce modern drugs and medicines.

Various schemes had been introduced to help people meet the high costs of medical services, all of them laudable, and every family in South Africa ought to be covered by one or other of these schemes. But for the successful operation of these schemes it was essential to secure the cooperation of the patient and the cooperation of the doctor.

The cooperation of the medical profession was needed and he appealed to the medical profession in South Africa to draw up a tariff for all medical treatments, just as the charges were specified in the tariffs of the medical aid societies. 'We know the medical profession,' he said. 'They are people of high repute who follow a noble profession and I know that one can appeal to such people.'

PASSING EVENTS : IN DIE VERBYGAAN

Southern African Cardiac Society (*Cape Province Section*). There will be a scientific and a business meeting in the E-floor lecture theatre, Groote Schuur Hospital, Observatory, Cape, on Thursday 12 March at 8·15 p.m. All interested are invited to attend.

* * *

Research Forum; University of Cape Town. A meeting of Research Forum will be held on Tuesday 17 March at 12 noon in the large A-floor lecture theatre, Groote Schuur Hospital, Observatory, Cape. Dr. J. A. H. Campbell will speak on 'Hepatic, pancreatic, and vessel changes following lower nephron nephrosis in rats'. All interested are invited to attend.

* * *

The South African National Council on Alcoholism. The 1959 Conference of this Council will be held in Cape Town from 27 to 29 April in the New Science Lecture Theatre, University of Cape Town. The Council hope to organize a series of popular seminars on the lines of a 'brains trust' and also to hold a special session for the medical profession. Further details will be published as soon as they become available.

* * *

Dr. Maurice Weinbren retired from his private radiotherapy practice in Johannesburg on 1 February 1959. In future, he will confine himself to his work at the Chamber of Mines Hospital, Johannesburg. Dr. Max Shapiro has combined his practice with that of Dr. A. H. Henning, who took over Dr. Weinbren's practice. They will continue their radiotherapy practice at Lister Buildings and the Princess Nursing Home, Johannesburg, and at Vereeniging.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South

Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association can only result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as to those who change their addresses within the Union.

* * *

Easter Stamp Fund. The National Council for the Care of Cripples in South Africa is again organizing the sale of Easter stamps. The proceeds of this annual effort enable the National Council and the local Cripple Care Associations to continue their work amongst cripples in South Africa. Practitioners engaged in the preventitive, curative and rehabilitative fields of cripple care recognize that the work of these voluntary organizations is an essential part of the national campaign to prevent crippling. The important work of the associations can only be continued if they receive adequate financial support from the public.

* * *

Mr. Michael W. Perrin, C.B.E., F.R.I.C., Chairman of The Wellcome Foundation, will be touring the Union from 25 February to 19 March. Mr. Perrin will visit the main centres of the Union and hold discussions with health and veterinary departments, universities, research organizations and members of Parliament. In addition to his duties with The Wellcome Foundation in Britain, Mr. Perrin is Hon. Treasurer of the Chemical Society and of the Parliamentary and Scientific Committee. He is chairman of the Institute of Cardiology in the Postgraduate Medical Federation of the University of London and a member of the Board of Governors of the National Heart Hospital, while his experience in the application of research has led to his appointment to the Board of Governors of the Chelsea College of Science and Technology, one of the recently recognized colleges of applied technology in Britain.

* * *

The World Medical Association. 7—10 May 1959 has been designated as Medicine Week by the Philippine Medical Association, which has united the efforts of 15 medical and allied organizations in planning and sponsoring its theme 'A Healthy Philippines Always'. The programme is planned to focus medical and public attention on the value of health and the importance of providing a scientifically sound medical programme in order to raise the standards of public health care for the nation's people.

The 15th General Assembly of the Japan Medical Congress will be held in Tokyo on 1-5 April 1959. The scientific session is under the joint auspices of the Confederation of the Medical Societies of Asia and Oceania and the Japan Medical Congress.

The Brazilian Medical Association has postponed its 2nd Congress, originally scheduled for the first week of September, to convene from 26 September to 4 October 1959. The theme of the Conference is 'Improving Medical Care Standards'. The dates of the meeting were changed so that delegates to the Second World Conference on Medical Education (Chicago, USA, 30 August-4 September 1959) and the 13th General Assembly of The World Medical Association (Montreal, Canada, 7-12 September 1959) would be able to attend the Brazilian Conference in Sao Paulo.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

ETISUL

Imperial Chemical Industries (S.A.) Ltd., introduce Etisul, a new drug for the treatment of leprosy, and supply the following information and comments:

Details of this new drug, Etisul, which may radically cut treatment time for leprosy, have been announced at the 7th International Congress on Leprology in Tokyo. The news was given to the Congress by Dr. T. F. Davey, head of the Nigerian Leprosy Service Research Unit at Uzuakoli, Eastern Nigeria, who has carried out clinical trials with the drug since April 1957.

A remarkable quality of the drug is that it is effective when administered by inunction. Etisul is in fact the only example in modern medicine of an anti-bacterial drug which can act throughout the body after rubbing into the skin. Etisul has not been in use long enough to allow categorical statements to be made about its value. It can, however, be said that hopes for the drug have been strengthened by the clinical results so far achieved.

The drug is easy to administer; anyone, unless he is gravely enfeebled, can rub the contents of a small tube into the skin until they disappear. From the leper's point of view application of treatment to the skin is psychologically satisfying. Etisul appears to have no toxic side-effects. It is hoped that treatment with Etisul will cut down very appreciably the length of time for which a patient need stay in a leprosarium. With most of the treatments available it may be anything up to 2 years before the patient can safely be discharged. With Etisul that period might well be cut down to about 3 months. This does not mean that the patient is cured in 3 months; it means that he no longer constitutes a danger to his fellows or his family and can be allowed to mingle freely with them and that he is no longer infective. This would mean that with little extra expense the existing leprosaria could handle many more patients than they do.

Moreover, the shorter the time for which a patient has to be separated from his family, the more readily will he come for treatment; and the more readily he comes for treatment, the more likely is he to come early. Not only are the results of early treatment better and quicker, but there is also less chance of infecting other people. Etisul might well, therefore, hasten the day when leprosy will disappear altogether.

PHASEÏN AND PHASEÏN FORTE

Noristan Laboratories (Pty.) Ltd. introduce Phaseïn and Phaseïn Forte and supply the following information:

Composed of reserpine and orphenadrine, which supplement and counteract each other, Phaseïn and Phaseïn Forte incorporate all the psychiatric facilities of reserpine treatment. At the same time the untoward side-effects of reserpine are eliminated. The action is more stabilized, avoiding the phase of turbulence, and these drugs may actually be referred to as harmonizers, because they restore mental equilibrium. They are much safer and easier to use in general practice than reserpine or phenothiazine derivatives.

Orphenadrine is a synthetic substance which has an anticholinergic action and atropine-like central effects. Owing to the orphenadrine content, none of the somatic or psychic side-effects of reserpine, such as constipation, nausea, vomiting, loss of initiative, and depression are generally experienced.

Reserpine and orphenadrine have a synergistic action. Therefore 0·2 mg. reserpine, as taken in Phaseïn, is equal in effect to 1-2 mg. reserpine taken alone.

Composition. Phaseïn is composed of 0·2 mg. reserpine and 20·0 mg. orphenadrine and Phaseïn Forte is composed of 1·0 mg. reserpine and 50·0 mg. orphenadrine.

Indications. Phaseïn is indicated in minor psychic disorders such as nervousness, neuroses and mental exhaustion. Phaseïn Forte is indicated in exogenous and endogenous psychoses, schizophrenia, and manic depressive psychoses.

Packings. Phaseïn and Phaseïn Forte are available as coated tablets in bottles of 20, 50 and 250.

Further particulars can be obtained from the South African Manufacturers, Noristan Laboratories (Pty.) Ltd.

PENITRIAD

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of Penitriad, a combination of penicillin VK and triple-sulphonamides for oral administration, and supply the following information. Each tablet contains phenoxymethylpenicillin potassium equivalent to 60 mg. of the free acid, sulphadimidine 167 mg., sulphadiazine 167 mg., and sulphathiazole 167 mg.

Penitriad is indicated in the treatment of infections due to penicillin and sulphonamide-sensitive organisms, including pneumococci, streptococci, *Staphylococcus aureus* and sensitive Gram-positive bacilli. In Gram-negative bacillary infections due to organisms of limited sensitivity to either penicillin or to sulphonamides, the activity of the combination is likely to be appreciably greater than that of either penicillin or a sulphonamide alone, notably in respiratory tract infections associated with the presence of *Haemophilus influenzae*.

The average adult dose is 2 tablets initially, followed by 1 or 2 tablets at 4-6 hourly intervals.

BOOK REVIEWS : BOEKRESENSIES

MENTAL ILLNESS

Bridging the Gap. From Fear to Understanding in Mental Illness. Edited by R. F. Tredgold, M.D., D.P.M. Pp. 270. 30s. net. London: Christopher Johnson Publishers Ltd. 1958.

In the past an atmosphere of hush-hush shrouded all happenings in mental institutions. Now the pendulum has swung over to the other extreme and we have over-publicity, an equally unhealthy situation. Recently some ex-patients and their friends broadcast stories of incidents at mental institutions in Britain which aroused considerable public interest and consternation.

It was alleged that people were illegally or recklessly committed to mental homes, detained without just cause and often deliberately subjected to exquisite cruelties. The medical and lay controllers of these institutions countered by stating that these tales were devoid of foundation or grossly exaggerated, and represented the outpourings of paranoid patients and of patients whose obsessions dominated and coloured all their thoughts and actions. But the public preferred to listen to the sensational hair-raising narratives of the ex-patients rather than the bland unadorned and unpalatable explanations of the doctors.

The book under review, compiled by men with first-hand acquaintance with mental hospitals, embodies a careful analysis of these stories and allegations, as well as a sympathetic consideration of the sweeping generalizations of both sides. While pronouncing unbiased and logical judgment, it makes constructive recommendations for bridging the gap between the attitude of the patient and the person entrusted with his welfare, and shows how mutual trust and cooperation can be substituted for fear and suspicion.

The book deals with conditions in Britain which have much in common with South African conditions and, therefore, anybody desirous of understanding and improving the care of psychiatric and senile patients in this country, would do well to study it.

I.M.H.

CLINICAL ENZYMOLOGY

Klinische Enzymologie. Die Fermente in der Pathogenese, Diagnostik und Therapie. Von Priv.-Doz. Dr. R. Abderhalden. viii+390 Seiten. 34 Abbildungen. DM. 48.00. Stuttgart: Georg Thieme Verlag. 1958.

At first the author gives a short introduction into enzymology. There he mentions definitions, terminology, chemical structure, methods for estimation of the activity, etc. After the introduction he describes several enzymes such as amylase, acetylcholin esterase, phosphatases, lysozym, insulinase, katalase, peptidases, lipase, etc. He deals with the physiological actions of the enzyme, with methods for determination of the ferment and with clinical findings. The next chapter concerns congenital diseases which are caused by ferments. Cystinosis, cystinuria, glycogenoses, lipoidoses, gout, adrenogenital syndrome, oxalosis and many other diseases are described. The last chapter is devoted to therapy by enzymes. Therapeutical actions of hyaluronidase, trypsin, streptokinase, plasmin and other ferments are mentioned. The book gives an excellent review of this field of research and can be recommended to everybody who is interested in enzymology or in most recent trends of medical research. A bibliography of 46 pages makes it possible to find the original articles.

H.W.W.

CORRESPONDENCE : BRIEWERUBRIEK

A POSTGRADUATE MEDICAL SCHOOL AT BLOEMFONTEIN

To the Editor: I hope you will allow me some space in the *Journal*, on behalf of the planning Committee of the proposed Postgraduate Medical School at Bloemfontein, to emphasize certain aspects of the project.

The Committee is indeed grateful to you for your Editorial[1] in the issue of the *Journal* of 3 January, which was most encouraging. The commentary[2] by Mr. T. B. McMurray, Hon. Registrar of the College of Physicians, Surgeons and Gynaecologists of South Africa, in the same issue, was also much appreciated.

With regard to 'the statistics of the National Hospital appearing meagre' it must be stressed that within a few years the number of beds will be increased to about 1,100 by the addition of a new non-European hospital of 300 beds. The Isolation Hospital of 216 beds was mentioned in our Memorandum and a very large mental hospital is also situated in Bloemfontein.

We fully realize that it will be a long time before our aims are achieved. After all, it took a long time before the Postgraduate Medical School was established in Great Britain, but since its inauguration at Hammersmith it has fully justified its existence. It is the considered opinion of the head of that institution that a postgraduate medical school, in order to give its staff untrammelled scope in research and the effective instruction of postgraduates, should be divorced from any medical school for undergraduates.

Unfortunately there is always a sort of vicious circle militating against the speedy realization of any great project. We are told, for instance, 'you haven't got the material or the men to carry out the functions of such a college'. On the other hand, before such an institution is actually established and has had time to build up a reputation, it will be difficult to attract sufficient numbers of students or men of eminence to give them instruction. Such arguments could go on *ad infinitum*. We must, however, 'cut the Gordian knot' and make a beginning or else nothing will ever be achieved.

We have the material to make a start. Furthermore, we have the wholehearted support of the authorities of the University of the Orange Free State, who are hampered only by financial considerations. It is already certain that the Provincial Administration will give all the support in their power towards the establishment of the postgraduate school. We feel convinced that, in the near future, adequate facilities for study and instruction in the 'basic sciences' will be provided.

With regard to the provision of adequate instruction for postgraduates, both theoretical and practical, we already have men in practice in Bloemfontein and on the hospital staff who have had experience in the instruction of undergraduates and who have the ability to teach postgraduates. The great success of refresher courses for general practitioners held here during the past 12 months, has been a good pointer and a great encouragement to the Planning Committee. It is hoped that in future men of greater eminence might be encouraged to come here, if at first only on a temporary basis. Later on it may confidently be assumed that, whatever the personnel, they will attain sufficient prestige to ensure that the institute shall grow from strength to strength.

In the meantime one of the first objects we wish to attain is fuller recognition by the South African Medical and Dental Council of work done at the National Hospital by aspirant specialists in most of the present specialities. The fact that only one out of two or more years' work is now recognized for specialist registration, is an apt illustration of how the vicious circle works. Without sufficient recognition it is impossible to attract sufficient numbers of postgraduates to staff the hospital adequately, or to encourage the part-time or honorary staff to spend more time in giving instruction.

The cutting of the vicious circle at this point has not yet been accomplished, but we are assured of a good deal of sympathetic support and encouragement from the Council.

Raymond Theron
Chairman of the Planning Committee
27 S.A. Mutual Building
Bloemfontein
17 February 1959

1. Van die Redaksie (1959): S. Afr. Med. J. 33, 3.
2. McMurray, T. B. (1959): *Ibid.*, 33, 15.